PESTICIDES ABSTRACTS

June 1975 - Vol. 8, No. 6

Abstracts 75-1235-1494

PSTAB 08(06) 335-431 (1975)

Quarterly Index Issue

U.S. ENVIRONMENTAL PROTECTION AGENCY

U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF PESTICIDE PROGRAMS
TECHNICAL SERVICES DIVISION
Rm. EB-49, 401 M Street, S.W.
Washington, D.C. 20460

PESTICIDES ABSTRACTS (formerly Health Aspects of Pesticides Abstract Bulletin) is a monthly publication of the Technical Services Division of the Office of Pesticide Programs, Environmental Protection Agency.

The purpose of this publication is to foster current awareness of the major worldwide literature pertaining to the effects of pesticides as defined in the Table of Contents. The publication represents a monthly review of more than 2,000 primary and secondary sources. A list of periodicals from which articles have been abstracted appears annually in the January issue. Subjects and authors of abstracted articles are cross-referenced in quarterly and annual indexes. Reprint requests should be directed to the author of the complete article.

Publication of an abstract, annotation, or citation does not imply endorsement by the Environmental Protection Agency of the original article or of commercial materials, equipment, or processes mentioned therein.

CONTENTS

Volume 8 June 1975 No. 6

Page

General 335
(News items, letters, and editorials appearing in scientific and technical journals; information on production and use; and other items of general interest)

Monitoring and Residues 338
(Levels of pesticides in people or nonexperimental animals or in water, air, soil, food, feed, and other consumer products)

Epidemiology, Prevention and Treatment 350
(Frequency and distribution of pesticide poisoning in man or nonexperimental animals and treatment of clinical cases; morbidity and mortality; safety measures; decontamination; disposal of containers and wastes; legislation and regulations)

Toxicology and Pharmacology 357
(Toxicity studies in man and animals; effects on cells, organs, and systems; metabolic studies, biochemistry, interactions; antidotes and other treatment; mode of action. Ordinarily, articles dealing with efficacy studies are not included.)

Analysis 385
(Preparation of samples; analytical methods; instrumentation)

Quarterly Index, January-March

Preface 394

Subject Index

I. Concepts 395

II. Compounds 406

Author Index

I. Personal 423

II. Corporate 431

75-1235. Golberg, L. (Inst. Comparative and Human Toxicol., Albany Med. Coll., Albany, NY). **Scope for innovation in teaching toxicology.** *Am. J. Pharm. Educ. 38(3): 354-362; 1974.*

The problem in teaching toxicology is how to inculcate and develop the critical perspective, the ability to comprehend and synthesize, which is needed to arrive at informed judgments on issues of toxicity, hazard, and safety. Research training should, to the greatest extent possible, reflect the practical application of the multidisciplinary approach. A course outline is provided which includes comparative embryology and teratology, morphology in toxicology, advanced principles of toxicology, and current issues in environmental toxicology. In the latter course, pesticide-related issues play a major pedagogic role.

75-1236. Kendrick, J. B., Jr. (Agric. Exper. Stn, Univ. California, Davis, CA 95616). **Pesticides and work safety.** *Calif. Agric. 29(1): 2; 1975.*

The Division of Agricultural Sciences at the University of California is engaged in a research project designed to develop better criteria for setting the waiting period that must be observed between application of a pesticide and re-entry into the field by workers who will come into contact with the treated plants. The study will determine how much pesticide goes onto the plants or elsewhere in the environment; what residues are left after various periods of time; and what physiological effects, if any, can be detected in workers who enter the fields after the official re-entry period. It is stressed that studies such as this one demonstrate the value of universities as public resources, for only in such institutions can so many diverse professional competencies be assembled to help solve these complex problems.

75-1237. Rawls, R. L. **Herbicide use rising but supplies remain short.** *Chem. Eng. News 53(11): 8-9; 1975.*

Most major herbicides are in short supply. There are two basic reasons for the inability of the industry to meet current demands. First, many companies do not have the necessary plant facilities. Second, some of the raw materials, among them hydrogen cyanide, are still hard to secure. However, the tightness of the market is viewed as a transitory condition. Most manufacturers are producing more herbicides this year than they did last. Thus farmers are expected to have a somewhat easier time in locating the products they need. Some materials have become more readily available, including chlorine/caustic soda and ethylene oxide. Some of this increased production traces to increased capacity. A drop is also expected in new farm acreage. Despite current production limitations, herbicides are the fastest-growing portion of the pesticide market and account for more than half of all dollar receipts in the pesticide industry.

75-1238. Dafter, R. (Author address not given). **Chemicals and pest control.** *Chem. Ind. (London) (18): 714-715; 1974.*

The concern and pressure for safety in the manufacture and production of pesticides, herbicides, and fungicides has caused the agrochemicals industry to be regarded as a high risk venture. The development of new products takes several years, and costs several millions of dollars. Some manufacturers are requesting that the patent life be extended to take account of this long development period. In recent years the market has continued to grow; the prospects for expansion are great. Major British companies in the field must be internationally minded, as the market within the United Kingdom is small in comparison with the world market. Britain does not have the severe climate, pernicious insects, or ravaging diseases which affect other nations, but it has a need for intensive agriculture in which crop protection chemicals are an important part. The view of this industry as a protector rather than a disturber of nature and ecology is presented.

75-1239. Visacki, V. (Yugoslav. Institute Technol. Belgrade, Yugoslavia). **Pravilnik o maksimalno dozvoljenim kolicinama pesticida u zivotnim namirnicama u sklopu resvanja problematike pesticida. [Rules on the maximum allowed quantities of pesticides in food articles and pesticides problems to be solved.]** *Hrana Ishrana 13(11-12): 571-573; 1972.* (3 references) (Serbo-Croatian)

Excessive pesticide residues have been detected in foods of animal origin due to improper pesticide application by inadequately trained commodity growers. Thus it is concluded that there is a need for adequate legal provisions regulating the production, transport, and application of pesticides. Current rules and regulations in Yugoslavia make efficient protection of the consumer impossible.

75-1240. Crosson, P. R. (Latin Am. Prog. for Resources for the Future, Inc., 1755 Massachusetts Ave., N. W., Washington, D. C. 20036). **Environmental considerations in expanding agricultural production.** *J. Soil Water Conserv. 30(1): 23-28; 1975.* (10 references)

The environmental problems associated with land conserving technologies resulting from the use of pesticides are discussed. Concern with pesticides in the environment centers on unintended damage to plant and animal life and to human health, and on the potential harm to the genetic stock of the race. There are known cases of pesticide poisoning of humans; of unintended elimination of natural insect predators; and of accumulation through the food chain of persistent pesticides, particularly in fish and birds, with resulting damage to reproductive processes. The use of DDT is banned in the United States and the use of aldrin and dieldrin is suspended. Parathion and other organophosphorus com-

pounds are bein substituted for DDT and other chlorinated hydrocarbons as insect controls. Application of organophosphorus pesticides has increased rapidly over the last decade even though the toxicity to mammals of some organophosphorus compounds, especially the parathions, is high. The most notable danger to man in the use of pesticides is the danger of accidental poisoning during application.

75-1241. Train, R. E. (Environ. Protect. Ag., Washington, D. C. 20460). **EPA and agriculture: establishing a partnership.** *J. Soil Water Conserv. 30(1): 33-35; 1975.*

The relationship between environmental protection goals and agricultural production is discussed. Agricultural producers are urged to develop a pesticide program that prevents overapplication, wind drift, and spraying of water bodies or non-target land areas, and avoids application during wind, rain, and other unfavorable conditions. The massive environmental damage that is inflicted in rural areas by pesticides also increases hazards to man and the food chain. Two Federal laws were established to protect the environment from pesticides. The 1972 amendments to the Federal Insecticide, Fungicide, and Rodenticide Act establish positive regulation to protect human beings and their environment and extend the rules to intra-state commerce. They prohibit registry of sale of a pesticide unless its labeling is adequate to prevent injury to man and damage to the environment. One of the purposes of the Federal Water Pollution Control Act of 1972 was to keep pesticide residues from contaminating the waterways. Analytical methods are being developed for predicting the effects and ultimate fate of agricultural chemicals after their application.

75-1242. Shindo, N. (Tokyo Org. Chem. Ind. Ltd., Tokyo, Japan). **The development of herbicides in Japan.** *Jpn. Pest. Inform. 22: 5-11; 1975.*

Insecticides account for 40.2% of all agricultural chemicals produced in Japan, herbicides 29.6%, fungicides 27.3%, and others 2.9%. The most popular type of herbicide formulation for paddy field use is the granule. A brief history of herbicides used in Japanese paddies is presented along with a list of herbicides currently being used in that country. The study of new herbicides in Japan has mainly centered on herbicides for use in paddy fields. About 20 new herbicides have been registered. The foreign trade in agricultural chemicals in Japan has become increasingly active year by year. Many herbicides are imported from the United States and Switzerland. The major herbicides exported from Japan include propanil, benthiocarb, linuron, maleic hydrazides, and sodium chlorate. About 20 kinds of important herbicides have been produced in Japan under license agreements with foreign companies. There has been a considerable increase in the number of Japanese pesticide patents recently, and the country is now one of the most advanced in this field of research. A new herbicide, NK-049 or 3,3′-dimethyl-4-methoxybenzophenone, has been made; this seems to be easily decomposed under natural conditions and has effective herbicidal action in rice paddy fields. However, since its effect is mild and the residue period is rather short, attempts are being studied to combine it with another herbicide. Future problems and prospects of herbicides are discussed.

75-1243. Matthysse, J. G. (Entomology Dep., Cornell Univ., Ithaca, NY). **Insecticides used on dairy cattle and in dairy barns: toxicity to man and cattle, hazards to the consumer and the environment.** *N. Y. State Assoc. Milk Food Sanit. Annu. Rep. 47: 15-31; 1973.* (49 references)

The toxicity and hazards of insecticides used on dairy cattle and in dairy barns are discussed. Pesticides that cause significant residues in milk must not be used on dairy cattle or in dairy barns. Since the cow rapidly excretes pesticides from her circulating blood into milk, only pesticides that are metabolized rapidly and are safe to mammals may be used. Rapidly metabolized or degraded organophosphorus compounds are used, such as crotoxyphos, dichlorvos, naled, and trichlorfon; or botanicals such as pyrethrins and their very low toxicity synergists or repellents. The insecticides are discussed accord to their method of use; toxicity to cattle, laboratory rats, birds, fish and wildlife; degradation time; tolerance in milk; and hazard to the consumer of dairy products. Only a small amount of these insecticides enters soil and water following proper use on dairy farms.

75-1244. Przybylski, Z. (Inst. Plant Protection, Rzeszow, Experimental Station Poznan, Poland). **Polichlorodwufenyle ich obecnosc w srodowisku naturalnym i wlasciwosci toksyczne. [Polychlorinated biphenyls, their presence in the environment and toxic properties.]** *Postep. Nauk Roln. 21/16 (5/148): 43-49; 1974.* (28 references) (Polish)

In a discussion and review it is stressed that agriculture is frequently blamed unjustly for the ubiquitous presence of toxic substances. A case in point is the presence of polychlorinated biphenyl compounds (PCB), which are frequently detected on chromatograms when testing for insecticides. PCBs, because of their physicochemical properties, are widely used in a variety of industries, especially the building, electrical, and chemical industries. Residues have been detected in a variety of fish, water fowl, and birds of prey, and egg shells. They have even been detected in human milk. It is possible that PCB is synthesized in nature through condensation of aromatic compounds in the presence of metals. They dissolve in water from dumped plastics and can penetrate into food products from plastic packaging. Because of their proven toxicity for experimental

animals, great stability in water, affinity for fats, and slow elimination from the body PCBs present a serious health problem requiring more and vigorous research.

75-1245. Lande, S. S. (Allegheny Country Health Dep., Pittsburgh, PA 15224). **Public attitude toward pesticides. A random survey of pesticides use in Allegheny County, PA.** *Pub. Health Rep. 90(1): 25-28; 1975.* (8 references)

A survey was conducted to determine public attitudes on the use of pesticides. The sites at which the survey was conducted were divided into categories and included: single-family dwellings, 41; commercial and recreational lawns, 7; institutions, 5; farms, 4; rights-of-way, 12; and wilderness and wasteland, 42. The conclusions of the study concurred with previous reports that the general public usually purchases and uses pesticides based on information supplied by the pesticide industry. The general public seemed satisfied with this situation and no more concerned about pesticides in their homes than about other household chemicals. Householders usually limited pesticide use to an aerosol or a lawn care herbicide. With the exception of golf courses, a farm and a nursery, and right-of-way maintenance programs, the majority of pesticides used consisted of low toxicity and low persistence chemicals. It was concluded that the pesticides used were not regarded as unusually dangerous either among all household chemicals or among all chemicals present in urban environments.

75-1246. Pimentel, D.; Goodman, N. (Dep. of Entomology and Section of Ecology and Systematics, Cornell Univ., Ithaca, NY). **Environmental impact of pesticides.** *IN: Survival in Toxic Environments, M. A. Q. Khan and J. P. Bederka, Jr., eds., Academic Press, New York, 1974, p. 25-52.* (18 references)

Evidence on the diverse effects of pesticides (insecticides, herbicides, nematocides, and fungicides) on the ecosystem is summarized with emphasis on direct and indirect effects. Certain current pesticide usage methods cause measurable damage to many species of birds, fish, and beneficial insects. Pesticides have influenced the structure and function of ecosystems and communities; reduced species population numbers in certain regions; altered the natural habitat under some conditions; changed the normal behavioral patterns in plants; increased or decreased the reproductive capacity of animals; altered the nutritional content of foods; increased the susceptibility of certain plants and animals to diseases and predators; and changed the natural evolution of species populations in some regions. Pesticide interactions may influence the toxic effects of pesticides upon species, resulting in either decreased toxicity or increased toxicity. Environmental stresses can also alter the susceptibility of animals to certain pollutants.

75-1247. Vettorazzi, G. (Food Additives Unit, W.H.O., Geneva, Switzerland). **The role of the World Health Organization in pesticide research.** *Toxicology 4(1): 31-40; 1975.* (30 references)

The activities of the World Health Organization in pesticide research are described. They relate to pesticide chemicals in such ways as toxicological evaluation of pesticides used in agriculture, recommendation of limits for pesticide residues in foods, evaluation of pesticides used in public health, and detection of resistance to pesticides. The Organization has directly sponsored research projects according to its limited budgetary capabilities, and assists in the promotion, guidance, and coordination of research programs on a world-wide basis. Specific projects include: estimation of the potential pesticide residue intake; effect of nutritional status on the toxicity of pesticide residues; effects of exposure to pesticides on the health of the general population; and evaluation and testing of new insecticides. There are a number of programs centering on the establishment of an acceptable daily intake (ADI) for pesticide chemicals.

75-1248. Kovacicova, J.; Batora, V. (Inst. Agric. Technol., Bratislava, Czechoslovakia). **Rezidua fenitrotionu a fosmetu m plodinach po aplikacii v roznych formach upravy. [Fenitrothion and Phosmet residues in crops treated with different formulations.]** *Agrochemia (Bratislava) 13(12): 349-351; 1973.* (12 references) (Czech)

Fenitrothion (in the form of emulsifiable concentrates, wettable powder, and aerosol preparation for aerial application) was tested under field conditions in sown lucerne, cereals, rape, apple, and pear trees. Relatively high levels of fenitrothion are found, particularly in lucerne, even 13 days after aerial application of the aerosol (29.9 mg/kg) or of the wettable powder (4.9 mg/kg). The results of the analysis of the other crops indicate amounts not in excess of 0.7 mg/kg which is the suggested tolerance in Czechoslovakia. Results on the persistence of phosmet in apples after the application of the emulsifiable preparation and the wettable powder indicate that there are no apparent differences between the preparations and the applied concentrations (0.1 and 0.05% A). Since the residues are relatively low, it can be expected that their toxicological interpretation will be favorable. The determination of fenitrothion was based on gas chromatography using CsBr–TID and that of phosmet on colorimetric enzymatic analysis.

75-1249. Huschenbeth, E. (Inst. Kuesten- und Binnenfisch., Bundesforschungsanst. Fisch., Hamburg, Germany). **Zur Speicherung von chlorierten Kohlenwasserstoffen im Fisch. [Accumulation of chlorinated pesticides in fish.]** *Arch. Fischereiwiss. Beiheft 24(1-3):* (7 references) (German)

Analyses were carried out on 2250 fishes, crustaceans, and bivalves to determine the residue accumulation of the pesticides DDT, TDE, DDE, lindane, and dieldrin. Most of the North-Atlantic, North-Sea and Baltic Seas lean fish investigated had accumulated less than 0.1 ppm of DDT and metabolites. In fat fish, in agreement with the fat-solubility of these substances, higher values were found. However, most of them did not exceed 1.0 ppm. Higher values were found only in salmon. The PCB values in the North Sea reached 0.2 ppm in lean fish, and 1.3 ppm in fat fish. The few freshwater fishes of the Moselle, Elbe, Aller, and Trave Rivers investigated in some cases showed a considerably higher contamination by DDT and its metabolites, with some values coming up to 6 ppm. However, most had accumulated less than 1.0 ppm. Here, also, the contamination by PCB's was slightly higher. Cod liver, containing much fat, naturally accumulates contaminants. That from the central Baltic contained up to 57 ppm total DDT, and even average values were determined to be about 30 ppm. During the sterilization of cod-liver, a large decrease in the DDT content was observed and a large increase in TDE was seen together with a considerable total reduction of the total DDT content. It was found that total DDT in cod liver was reduced during cooling by more than 50%, but during frying by only about 10%.

75-1250. Fipipan, T.; Sjerobabski, D.; Musac, I. (Safety Inst. Zagreb, Yugoslavia). **Istrazinanje koncentracija aerosola pesticida za vrije'e zaprosirvanja poljoprivrednih kultura. [Investigation of aerosol concentration of pesticides during the treatment of agricultural produce.]** *Arh. Hig. Rada Toksikol. 25(2): 313-312; 1974.* (Serbo-Croatian)

Results are presented from investigations on aerosols from treatment-shed air samples taken during the treatment of agricultural produce with Pepein S-50 (active substance DDT) and on airborne dust during the treatment of seed with Radosan (active substance organically bound mercury). It was concluded that on many occasions the concentration of the active substances DDT and organically bound mercury are several times higher than the threshold limit values.

75-1251. Cencelj, J.; Dorer, M. (Inst. Agric. Slovenia, Ljubljana, Yugoslavia). **Ostanki pesticidov v tleh in vodah. [Residues of pesticides in soil and water.]** *Arh. Hig. Rada Toksikol. 25(2): 253-256; 1974.* (8 references) (Serbo-Croatian)

The degradation and accumulation of prometryne (a herbicide of the triazine group) in two different soil types, the rendzina and brown soil, were observed in pot experiments in the open without vegetation. In the summer season degradation of prometryne in the upper layer of rendzina (up to 5 cm) with pH = 7 and an average of 9% humus was 4 times slower than on brown soil with pH = 5–6 and a lesser amount of humus (6%). Migration of the lower layers (up to 15 cm) was more regular. However, after six months, the relative degradation of prometryne in brown soil was slower in the upper layers. The presence of chlorinated pesticide residues in water was determined on fish samples since these residues were especially accumulated in some parts of aquatic animals. The presence of DDT and its metabolites was established by analysis of fish samples from the Slovenian coast of the Adriatic. In some instances measurable quantities of dieldrin were also found.

75-1252. Picer, N.; Picer, M.; Strohal, P. (Cent. Marine Res., Ruder Boskovic Inst., Zagreb, Yugoslavia). **Istrazivanje procesa adsorpcije i desorpcije DDT-a na morskim sedimentima. I. Metodika i kineticka istrazivanja. [Investigation of processes of DDT adsorption and desorption on marine sediments. I. Methods and kinetic investigation.]** *Arh. Hig. Rada Toksikol. 25(2): 317-318; 1974.* (Serbo-Croatian)

The interaction of DDT with natural and treated marine sediments, limestone, and quartz was investigated by means of a radiotracer technique. Methods were

developed for ^{14}C labelled DDT counting by means of liquid scintillators in heterogeneous systems such as sea water and in mixtures of sea water with limestone, quartz, natural and treated marine sediments. The use of blanks in the calculation of DDT distribution beween liquid and solid phase in kinetic investigations is described. Results indicate that absorption processes in the sea water medium are very fast in the case of natural and treated sea sediments. Equilibrium is reached within one hour. The rate of DDT adsorption on limestone is slightly lower, while it is significantly lower in the case of quartz. To attain equilibrium distribution of DDT between sea water and quartz, mixing of the system for more than five hours is required.

75-1253. Picer, M.; Picer, N.; Strohal, P. (Cent. Marine Res., Ruder Boskovic Inst., Zagreb, Yugoslavia). **Istrazivanje procesa adsorpcije i desorpcije DDT-a na morskim sedimentima. II. Ravnotezna distribucija DDT-a izmedu tekuce i krute faze. [Investigation of processes of DDT adsorption and desorption of marine sediments. II. Equilibrium distribution of DDT between liquid and solid phase.]** *Arh. Hig. Rada Toksikol. 25(2): 318; 1974.* (Serbo-Croatian)

The equilibrium distribution of DDT between liquid and solid phases in sea water was investigated by means of a radiotracer technique. The influence of the mode of measuring DDT concentration in liquid and solid phases on the calculation of its equilibrium distribution between the two phases is discussed. The effect of DDT concentration on its adsorption in several solid phases was studied and the results were given as adsorption isoterms.Desorption experiments indicate that these processes are reversible. The extent of desorption is larger from quartz than from limestone, while it is significantly lower than from sea sediments. Examination of the equilibrium distribution of DDT between sea water and the surfaces examined suggested that DDT is present as a colloidal dispersion. Results are also discussed in terms of marine pollution.

75-1254. Regan, A.; Krempler, F. (Inst. Plant Protect., Zagreb, Yugoslavia). **Ostaci triazinskih herbicida u tlu. [Residues of triazine in the soil.]** *Arh. Hig. Rada Toksikol. 25(2): 319; 1974.* (Serbo-Croatian)

Triazine residues in soil were examined over a three-year period. Degradation of atrazine and simazine, applied at different dose levels, was examined at two soil depths in cornfields, orchards, and vineyards. Residues of atrazine and atrazine plus prometryne were also investigated after application of different formulations (wettable powder, microgranules and liquid). Residues were detected by GLC using a CsCl alkali flame ionization detector. Degradation was maximal during the first month after application of both atrazine and simazine, while atrazine degraded more rapidly during the following months. Two to three months after application, the concentration of atrazine and simazine was similar in the upper and lower layers, but degradation was slower in less thoroughly cultivated soil. No differences in atrazine and atrazine plus prometryne residues were apparent for the various formulations.

75-1255. Otaci, C.; Tuglular, R.; Turhan, K.; Ertugrul, G. (Anal. Lab., Inst. Agric., Istanbul, Turkey). **Marmara bolgesinde seftali zaralilarina karsi kullanilan ilaclardan gusathion'un bakiye analizleri. [Residue analysis of gusathion used against peach pests in the Marmara region.]** *Bitki Koruma Bul. 13(2): 73-82; 1973.* (1 reference) (Turkish)

The insecticides methyl gusathion (azinphosmethyl) 25% WP, gusathion A (azinphosethyl) 40% Em, and gusathion (azinophosmethyl) 20% Em were applied at the 0.01% level to Hale variety peaches on three dates. Results were taken only from the Odunluk region of Bursa in 1968, but in 1969 they were taken from both the Odunluk and Arabayatagi regions. Samples were collected two days before the last application and 1, 3, 7, 10, and 13 days thereafter, and residue analyses were carried out. According to the analyses, performed by the modified Meacher method, the residue levels after 13 days were: Odunluk 1968, 20% Em 0.44 ppm, 25% WP 0.46 ppm; Odunluk 1969, 20% Em 0.46 ppm, 25% WP 1.5 ppm (thought to have been the result of nonuniform application), 40% Em 0.75 ppm; in Arabayatagi 20% Em 0.26 ppm, 25% WP 0.52 ppm, and 40% Em 0.48 ppm. These results are below the US tolerance for fruit, which is 2 ppm, but are close to the West German tolerance of 0.5 ppm.

75-1256. Norris, L. A.; Montgomery, M. L. (U.S. Dep. Agric., For. Serv., Pacific Northwest For. Range Exp. Stn., For. Sci. Lab., Corvallis, OR 97331). **Dicamba residues in streams after forest spraying.** *Bull. Environ. Contam. Toxicol. 13(1): 1-8; 1975.* (16 references)

The concentrations of the herbicide dicamba were measured in streams flowing from forest lands in Hebo, Oregon which had received aerial application of herbicide (1.12 kg dicamba and 2.24 kg 2,4-D per ha in water). Analysis was carried out using a gas chromatograph. Dicamba residues were first found in the stream samples (taken about 1.3 km from where the stream entered the treatment area) about 2 hours after the start of application. Residue levels rose sharply to a maximum of 37 ppb in about 5.2 hours then declined slowly to background levels by 37.5 hours. Small additional amounts of herbicide entered the stream several days later, probably in connection with a coincident period of precipitation. No dicamba residues were found more than 11 days after application, even after heavy storms. In two other areas where opportunities for drift and direct application of spray were limited, no detectable dicamba residues were found at any time. Dicamba does not appear to be particularly mobile in the forest floor and

soil. The concentration of dicamba was markedly reduced with downstream movement over distances of 0.6 to 3.7 km from the original sampling point. Dicamba was apparently rapidly adsorbed by stream sediments and detritus or taken up by aquatic biota in the stream. Dicamba residues in this system appeared to pose no acute hazard to aquatic organisms or downstream water users and the short persistence of the herbicide precluded chronic exposure. The herbicide can therefore be used for brush control of forest lands with little or no impact on the aquatic environment if direct application to surface waters is minimized.

75-1257. Impson, J. W.; Epps, E. A., Jr.; Smilie, J. L. (Louisiana Coop. Extension Serv., Louisiana State Univ., Baton Rouge, LA). **Removal of DDT from sweet potato washwater.** *Bull. Environ. Contam. Toxicol. 13(1):37-39, 1975.* (1 reference)

To protect stored sweet potatoes from the sweet potato weevil, the potatoes are treated with DDT dust. When they are removed from storage, they are washed prior to grading and shipping; the DDT residues on the washed potatoes are usually no more than 1 ppm, but the washwater may contain enough pesticide to be a danger to aquatic biota. A method for the treatment of washwater to remove DDT involves the addition of lime to the water. The need to calibrate the addition of lime according to flow rate of the washwater prompted a re-evaluation of this process. Preliminary studies indicate that 3-6 lbs of hydrated lime per thousand gallons of water and 1-hour settling time would give good DDT removal. Full-scale testing indicated that, although the level of DDT removal at the 6-lb lime rate was high, the discharge still contained unacceptable levels of DDT. Only slight improvement was obtained with higher rates of liming and combining lime with ferrous sulfate. If reduction to less than 10 ppb DDT is necessary, larger stilling basins or use of a filter cloth or other filter medium would be necessary.

75-1258. Doguchi, M.; Fukano, S. (Tokyo Metrop. Res. Lab. Public Health, Shinjuki-ku, Tokyo, Japan). **Residue levels of polychlorinated terphenyls, polychlorinated biphenyls and DDT in human blood.** *Bull. Environ. Contam. Toxicol. 13(1): 57-63; 1975.* (13 references)

Blood samples from 19 male and 8 female volunteers from the Tokyo Metropolitan area were analyzed for residues of DDT, PCBs, and polychlorinated terphenyls (PCTs) using gas chromatographic mass spectrometry. The subjects, who ranged in age from 21-57 years, had no occupational exposure to PCBs or PCTs. All samples contained substantial quantities of PCTs, PCBs, and DDE, the concentrations averaging 5.0 ppb, 3.2 ppb, and 11.2 ppb, respectively. The data for DDE represented the DDE originally present in the blood as well as the *p,p'*-DDT present; the latter was converted to *p,p'*-DDE by alcoholic alkaline hydrolysis. The levels of DDT obtained in this study probably reflect the approximate levels of total DDT and metabolites in the samples.

75-1259. Dimond, J. B.; Owen, R. B., Jr.; Getchell, A. S. (Life Sci. Agric. Exp. Stn., Univ. Maine, Orono, ME 04473). **DDT residues in forest biota: further data.** *Bull. Environ. Contam. Toxicol. 13(1): 117-122; 1975.* (122 references)

Between 1966 and 1969, 54 frogs and tadpoles (*Rana clamitans, R. pipiens, R. palustris*, and *R. catesbiana*), 10 toads (*Bufo americana*), 24 garter snakes (*Thamnophis sirtalis*), and 6 ravens (*Corvus corax*) were collected from Maine forests sprayed with DDT (1 lb/acre) one, two, or three times between 1958 and 1967. Total body residues of DDT were determined using gas chromatography. No high residue levels were found in frogs, toads, or snakes, although data for year of treatment are lacking; estimated residues in the year of spray would be about 1 ppm for frogs and 2-3 ppm for tadpoles, toads, and snakes. Frogs, toads, and snakes do not therefore appear threatened by single or infrequent moderate applications of DDT. Residue levels in the ravens showed no relation to the DDT-treatment history of the collection sites. The residues in these birds were high, averaging well above any other species studied. Thus, ravens will accumulate high residue levels under conditions of moderate forest use of DDT, discontinued agricultural use, and use in rural villages.

75-1260. Cochrane, W. P.; Russell, J. B. (Anal. Serv. Sect., Plant Prod. Div., Canada Agric., Ottawa, Ontario, Canada). **Residues in wheat and soil treated with the mixed butyl esters of 2,4-D.** *Can. J. Plant Sci. 55(1): 323-325; 1975.* (9 references)

The decline of 2,4-D residues in both wheat and soil was monitored after treatment with a mixed butyl ester formulation. No ester residues were found in any wheat samples. Immediately following spray application, a level of 8.35 ppm 2,4-D acid was obtained. This gradually decreased to less than 0.01 ppm in 44 days. No residues were found in the harvested grain or threshed straw collected after 80 days. Recoveries of 2,4-D residues from fortified soil samples using acidic acetone or acidic ether extraction gave recoveries in the range of 81-95%, with a lower limit of detection at the 0.01 ppm level. From an initial low free 2,4-D acid residue of 0.0004 ppm, an increase occurred, which reached a maximum of 0.130 ppm at 8 days and then rapidly declined to less than 0.01 ppm within 22 days of application.

75-1261. Hruska, J. (Sanit. Stn., Ceske Budejovice, Czechoslovakia). **Rezidua DDT ve vejcich a tuku drubeze. [DDT residues in poulty eggs and fat.]** *Cesk. Hyg. 18(1): 33-40; 1973.* (10 references) (Czech)

Results are presented of DDT residues in poultry eggs and fat and explanations are given of the effects of technology in large scale agricultural production on the DDT content of eggs. It is noted that large-scale breeding is sometimes a source of heavy DDT contamination of market eggs and fattened poultry. On samples of eggs from fowl-run breeding, DDT residues were found in 66% of cases and the average content was 0.23 mg/kg. In large-scale indoor breedings 59% of samples were positive with the average level of the residues being 1.9 mg/kg. The frequency of DDT application correlates with the content found in the eggs. The average content of DDT accumulated in fat of indoor bred chickens was 3.6 mg/kg, with a maximum level of 39.7 mg/kg found in a shed where DDT was regularly applied.

75-1262. Halacka, K.; Vymetal, F. (Dep. Hyg., Sch. Med., VJEVP, Brno, Czechoslovakia). **Sledovani retence DDT a DDE v lidske tukove tkani v nekolikaletem casovem odstupu. [Observation of the retention of DDT and DDE in human adipose tissues after several years.]** *Cesk. Hyg. 18(8): 372-376; 1973.* (10 references) (Czech)

Investigation into DDT and DDE retention in subcutaneous fatty tissue was made on 350 persons of different age, sex and occupation in a selected region of Czechoslovakia, 1970/72. The values were 3.2 ppm ± 1.8 for DDT and 2.2 ± 1.6 for DDE. In a comparison with a previous examination in 1963/64 using the same analytical procedure, the values of DDT and DDE residues proved to be markedly lower (by 41.8% when calculated as a corrected sum (as DDT). As in 1963/64 there were no differences in relation to sex, geographic locale, occupation, or age. There was a certain correlation between the observed amount of DDT and its metabolites and the amount of DDT used in the agriculture of the observed area (which decreased by 35% during the intervening period). In view of the continuing potential health risk and general toxic situation a complete prohibition of DDT in Czechoslovakia is considered.

75-1263. Crisetig, G.; Viviani, R.; Mora, A. (Ist. Biochim. Fac. Med. Vet., Univ. Bologna, Bologna, Italy). **Residui di idrocarburi clorurati in carni bovine. [Chlorinated hydrocarbon residues in cattle meat.]** *Atti Soc. Ital. Sci. Vet. 26: 470-474; 1972.* (7 references) (Italian)

The amounts of chlorinated hydrocarbon residues in the adipose and muscular tissues of cattle are given. Maximum values in muscular lean and fat are: lindane 0.4-0.8 ppm; heptachlor epoxide 0.066-0.153 ppm; BHC 0.139-0.140 ppm; DDE 0.07-0.133 ppm; and dieldrin 0.037-0.05 ppm. It is also shown that, estimated via their lipid content, fat residues are correlated with those from muscles with a probability of error between 0.1 and 5%.

75-1264. McConnell, G.; Ferguson, D. M.; Pearson, C.. R. (Res. Dep., Imperial Chemical Industries, Ltd., London, England). **Chlorinated hydrocarbons and the environment.** *Endeavour 34(121): 13-18; 1975.* (27 references)

The environmental impact of major chlorinated aliphatic hydrocarbons is discussed. Chloroform, carbon tetrachloride, trichloroethylene, perchloroethylene, and trichloroethane are very widely distributed in the environment at a concentration level of 1 ppt. The concentrations of DDT and polychlorinated biphenyls in fatty tissues are at least three orders of magnitude greater than those of the industrial solvents. A population may breathe air, drink water, and consume food containing trace quantities of these materials. All these compounds are fairly rapidly degraded in the environment to carbon dioxide, water, and chloride ion. There is no evidence for their significant bioaccumulation, via the food chains, to higher trophic levels.

75-1265. Chian, E. S. K.; Bruce, W. N.; Fang, H. H. P. (Environ. Eng., Civil Eng. Dep., Univ. Illinois, Urbana, IL 61801). **Removal of pesticides by reverse osmosis.** *Environ. Sci. Technol. 9(1): 52-59; 1975.* (24 references)

Two types of reverse osmosis membrances—i.e., cellulose acetate (CA) and cross-linked polyethylenimine, designated as NS-100 by OSW, were evaluated for their removal of a wide variety of pesticides, including chlorinated hydrocarbons, organophosphorus, and miscellaneous pesticides. With each membrane the rejection of pesticides was better than 99%. A considerable amount of pesticide was adsorbed onto the membrane materials. The extent of adsorption is governed by the van der Waals–London forces and hydrophobic bonding between pesticide molecules and the polymeric membrane materials. Membrane rejection of the pesticides is in turn governed by the polarity of the solute molecules in aqueous solution, which is in accord with the theory advanced by Sourirajan. (Author abstract by permission)

75-1266. Fiedoruk-Poplawska, T. (Katedry Hig. Ogolnej, Bialystok, Poland). **Zawartosc DDT w wodach do picia wojewodztwa Bialostockiego. [The amount of DDT in drinking water of Bialystok Province.]** *Folia Med. Bialostocensia 1(1): 177-182; 1972.* (6 references) (Polish)

The DDT content of Bialystok province drinking water was determined. Samples were taken from various wells and in 55 cases increased concentrations of DDT were found (43.6% of the total number of wells studied). Also, during spring DDT content levels were higher than in autumn.

75-1267. Deputat, G. Ye. (Zhdanov District Sanit. Epidemiol. Stn., Moscow, USSR). **Sanitarnyy kontrol' za**

chuzherodnymi khimicheskimi veshchestvami v produktakh pitaniya. [Sanitary control of foreign chemical substances in food products.] *Gig. Sanit. 38: 89; 1973.* (Russian)

Food contamination with foreign chemical substances such as food additives and pesticides, and the current state of sanitary control is discussed. Both the pesticide residue levels in food products and the incidence of pesticide residues have decreased substantially during the 5 years from 1966 to 1970. While 62.5% of all food samples tested were free of pesticides in 1966, this percentage rose to 88% in 1970. The percentage of food samples with pesticide residues within the acceptable limits decreased from 15.5% in 1966 to 10.9% in 1970. Pesticide residues in excess of the maximum allowable level were found in 22% of all samples tested in 1966, and in only 1.1% of all cases in 1970.

75-1268. Kamata, T. (Sch. Hyg., Fac. Med., Hiroshima Univ., Hiroshima, Japan). **Hygienic studies on pesticide residues.** *Hiroshima Daigaku Igaku Zasshi (Med. J. Hiroshima Univ.) 22(9-10): 547; 1974.* (1 reference) (Japanese)

Hygienic studies of BHC residues have revealed that the differences in rates of accumulation for the 4 isomers of BHC are due to their different chemical stabilities, and that the α-, γ- and δ-isomers isomerize into the β-isomer in living organisms. Previous studies pointed out that the source of pollution of cow's milk by BHC was the rice straw fed to the cows, and the route of pollution was elucidated. Rather rapid disappearance of BHC from cow's milk is predicted as a result of the ban on insecticidal use of BHC in rice fields. It is also pointed out that while the pesticide residues in agricultural crops have in general been decreasing, appreciable and steady levels remain in fish, shellfish, human milk, and human tissues. (Doctoral thesis presented at the 180th lecture meeting of the Faculty of Medicine, Hiroshima Univ., Sept. 21, 1974)

75-1269. Yamada, T.; Sakamoto, Y. (Hiroshima Prefect. Inst. Pub. Health, Hiroshima, Japan). **[Survey of pesticide residues in vegetables, fruits, cow's milk, fish, and shellfish.]** *Hiroshima-Ken Eisei Kenkyu-Sho Fuzoku Kogai Kenkyu-Sho Gyomu Nenpo (Hiroshima Prefect. Inst. Prevention Environ. Pollut. Annu. Rep.) 47: 54-56, 1973.* (1 reference) (Japanese)

Examinations of 117 specimens of vegetables (19 species) and fruits (10 species) revealed that no specimen exceeded the ministry's standard in amount of any specific pesticide residue (i.e. Pb, BHC, DDT, aldrin, carbaryl, parathion, EPN, malathion, endrin and dieldrin). No organophosphorus pesticide residues were detected. The most frequently detected pesticide residue was BHC; the highest concentration was found in the case of Japanese radish at 0.019 ppm. The amount detected in 1973 was considerably lower than that found in 1971 and 1972. Results in 7 (1972) and 17 (1973) specimens of coconut milk show that the decrease from the 1971 level was great, and the amount of residue considerably lower than the Ministry's temporary standards. Results in 8 specimens of 5 species of surface and bottom dwelling fish and shellfish showed no difference in residues between surface and bottom fish, and lindane was detected in oyster and short-necked clam but not in sea eels and flatfish.

75-1270. Burns, J. E.; Miller, F. M. (Inst. Environ. Med., New York Univ. Med. Cent., New York, NY 10016). **Hexachlorobenzene contamination: its effects in a Louisiana population.** *Arch. Environ. Health 30(1): 44-48; 1975.* (13 references)

Plasma hexachlorobenzene (HCB) residues in a Louisiana population exposed through the transportation and disposal of chemical waste containing HCB averaged 3.6 ± 4.3 parts per billion (ppb) in a sample of 86 people. The highest level was 345 ppb in a waste disposal facility worker while the highest level in the general population was 23 ppb. There was no evidence of cutaneous porphyria by history or skin examination. Males had significantly higher residues than females (4.71 vs. 2.79 ppb) but there were no age differences. A possible association between HCB residues and coproporphyrin and lactic dehydrogenase was found. Only 2 of 48 meals sampled were positive for HCB. There was a correlation between HCB levels in household dust and plasma levels. Perchloroethylene and carbon tetrachloride production workers had plasma HCB levels of up to 233 ppb. (Author abstract by permission)

75-1271. Booth, N. H.; McDowell, J. R. (Dep. Physiol. Pharmacol., Coll. Vet. Med., Univ. Georgia, Athens, GA 30601). **Toxicity of hexachlorobenzene and associated residues in edible animal tissues.** *J. Am. Vet. Med. Assoc. 166(6):591-595; 1975.* (26 references)

The USDA has detected hexachlorobenzene (HCB) in the body fat of domestic animals and poultry in at least 14 states. In Louisiana, industrial contamination appeared to be largely responsible for the HCB residues detected in cattle. Following the episode in Louisiana, the Environmental Protection Agency established an interim tolerance of 0.5 ppm for HCB in the fat of cattle, sheep, goats, and horses. Edible meat products containing HCB at higher concentrations are violative and cannot be used as a source of food for human consumption. (Author abstract by permission)

75-1272. Adamovic, V. M.; Sokic, B.; Bojovic, B. (Zdrav. Saventik, Belgrade, Yugoslavia). **Organohlorni insekticidi kao merilo higijenske ispravnosti, porekla i nacina proizvodnje svinjske masti. [Organochlorine insecticides as a criterion of hygienic appropriateness,**

origin, and way of production of lard.] *Hrana Ishrana 13: 11-12; 1972.* (6 references) (Serbo-Croatian)

Results obtained from investigations concerning the contamination of lard by organochlorine insecticides from 1969 to 1972 are presented. The investigation involved several hundred samples representing thousands of tons of lard for human use in Yugoslavia. The lard originated from various countries (such as Bulgaria, Romania, U.S.A., etc), or was made in Yugoslavia. Results show that the level of contamination may be followed with reference to the time and place of production. Total BHC levels ranged from 0.05 to 0.2 ppm, and total DDT from 0.4 to 2.28 ppm. No definite increasing or decreasing trend was apparent for this three year period.

75-1273. Nakagawa, M.; Crosby, D. G.* (Dep. Environ. Toxicol., Univ. California, Davis, CA 95616). **Photonucleophilic reactions of nitrofen.** *J. Agric. Food Chem. 22(6): 930-933; 1974.* (31 references)

The sunlight photolysis of nitrofen (2,4-dichlorophenyl *p*-nitrophenyl ether) in aqueous methanol represents a photonucleophilic displacement of nitrophenate by the hydroxide ion of water. In the presence of potassium cyanide, irradiation of nitrofen resulted in 2,4-dichlorobenzonitrile and related compounds, while the photoreaction with piperidine provided *p*-nitrophenol as well as tarry products from the further reactions of intermediate 2,4-dichlorophenol with the amine. 2,4-Dichlorophenol also reacted with cyanide to form 2,4-dicyanophenol (4-hydroxyisophthalonitrile). These and other photonucleophilic reactions may help to explain the environmental dissipation of many pesticides. (Author abstract by permission)

75-1274. Schooley, D. A. Bergot, B. J.; Dunham, L. L.; Siddall, J. B. (Zoecon Corp. Res. Lab., Palo Atlo, CA 94304). **Environmental degradation of the insect growth regulator methoprene (isopropyl(2E,4E)-11-methoxy-3,7,11-trimethyl-2,4-dodecadienoate). II. Metabolism by aquatic organisms.** *J. Agric. Food Chem. 23(2): 293-298; 1975.* (16 references)

Biodegradation of isopropyl (2E,4E)-11-methoxy-3,7-11-trimethyl-2,4-dodecadienoate, a new insect growth regulator (common name, methoprene; trademark, Altosid), was studied in pond water containing unknown microorganisms. A time plot of recovery of (2E)-[10-^{3}H]methoprene from pond water showed a half-life of approximately 30 hr at 0.001 ppm and 40 hr at 0.01 ppm. Incubation of (2E)-[10-^{3}H]methoprene for 3 days at 0.42 ppm generated three primary metabolites, the result of ester hydrolysis and/or *O*-demethylation. These metabolites and recovered methoprene were photoequilibrium mixtures of 2-ene double bond isomers. In another incubation experiment with (2E)-[5-^{14}C]methoprene at 0.66 ppm in a pond water sample with presumably different microflora, a completely different metabolic profile was observed, the sole identifiable metabolite resulting from oxidative scission of the 4-ene double bond. The principal metabolite in the latter experiment was 7-methoxycitronellic acid (29% of applied dose). (Author abstract by permission)

75-1275. Quistad, G. B.; Staiger, L. E.; Schooley, D. A. (Zoecon Corp. Res. Lab., Palo Alto, CA 94304). **Environmental degradation of the insect growth regulator methoprene (isopropyl (2E,4E)-11-methoxy-3,7,11-trimethyl-2,4-dodecadienoate). III. Photodecomposition.** *J. Agric. Food Chem. 23(2): 299-303; 1975.* (16 references)

Photodecomposition products were characterized after irradiation of methoprene as a thin film on glass or silica gel, as an aqueous emulsion, and as a methanolic solution with added photosensitizer. The most abundant photoproduct of methoprene after illumination of a thin film or aqueous emulsion was 7-methoxycitronellal (9-14%). Methoprene was quite stable in methanolic solution although a slow reaction with singlet oxygen occurred. Isomerization of the 2-ene double bond to a mixture of cis-trans isomers was facile. (Author abstract by permission)

75-1276. Soderquist, C. J.; Crosby, D. G.; Moilanen, K. W.; Seiber, J. N.*; Woodrow, J. E. (Dep. Environ. Toxicol., Univ. California, Davis, CA 95616). **Occurrence of trifluralin and its photoproducts in air.** *J. Agric. Food Chem. 23(2): 304-309; 1975.* (20 references)

Trifluralin vapor photodecomposed to a number of products in a laboratory vapor-phase reactor which simulated sunlight conditions. For analysis, an air sampling method utilizing a coated solid adsorbent as a vapor trap was developed which trapped these materials at air flow rates greater than 1 m^3/min and with a limit of detectability of less than 1 ng/m^3. Trifluralin and several photoproducts were detected in the air above both surface treated and soil incorporated fields. These photoproducts probably arise primarily from photolysis of trifluralin on the soil surface followed by volatilization; however, the contribution of vapor-phase photolysis of trifluralin undoubtedly increases with increasing air residence time. (Author abstract by permission)

75-1277. Yu, C. C.; Booth, G. M.; Hansen, D. J.; Larsen, J. R. (Dep. Entomol., New York State Agric. Exp. Stn., Geneva, NY 14456). **Fate of pyrazon in a model ecosystem.** *J. Agric. Food Chem. 23(2): 309-311; 1975.* (12 references)

Pyridazinone ring-^{14}C-labeled pyrazon [5-amino-4-chloro-2-phenyl-3(2*H*)-pyridazinone] was slowly degraded in water. Thirty-two days after application of the compound to a model ecosystem, about 66% of the radioactivity in the water was found to be the parent

compound. Very small amounts of 2-dephenylpyrazon [5-amino-4-chloro-3(2*H*)-pyridazinone] and five other unknown spots (combined total 1%) were detected only after acid hydrolysis. The remainder of the radioactivity was present as unextractable water-soluble products (33%). Combined parent compound and metabolites in organisms living in the ecosystem ranged from 0.06 ppm in fish to 0.6 ppm in crab. Analysis of the crab extracts revealed that no 2-dephenylpyrazon was present and that the parent pyrazon constituted about 76% of the total radioactivity in that organism. There was no evidence to indicate that pyrazon and its degradation products were magnified through the food chain. (Author abstract by permission)

75-1278. Tappan, W. B.; Wheeler, W. B.; Lundy, H. W. (Agric. Res. Educ. Cent., Univ. Florida, Quincy, FL 32351). **Insect control and chemical residues after applying acephate on cigar-wrapper and flue-cured tobaccos in Florida.** *J. Econ. Entomol. 67(5): 648-650; 1974.* (4 references)

Acephate as a spray and dust was evaluated in 1971-72 for control of the green peach aphid, *Myzus persicae* (Sulzer), tobacco budworm, *Heliothis virescens* (F.), and tobacco hornworm, *Manduca sexta* (L.), on cigar-wrapper and flue-cured tobaccos. Season-long control of the 3 insects was effective with 6–7 applications at rates ranging from 0.72–1.11 lb AI/acre. In 1972, samples of green, cured, and fermented cigar-wrapper and cured flue-cured tobaccos were analyzed for residues of acephate and its metabolite, Ortho 9006 (*O,S*-dimethyl phosphoramidothioate), after application of acephate insecticide. Residues of acephate and Ortho 9006 on the green leaf were 15.7 and 1.3 ppm, respectively, 14 days after the last of 6 spray applications at 1.01 lb AI/acre. No residue of the 2 compounds was found on the cured leaf of either tobacco. This insecticide loss probably was caused by the elevated temperatures during the curing and fermentation processes. (Author abstract by permission)

75-1279. Sameoto, D. D.; Darrow, D. C.; Guildford, S. (Dep. Environ., Fish. Mar. Serv., Mar. Ecol. Lab., Bedford Inst. Oceanogr., Dartmouth, Nova Scotia, Canada). **DDT residues in euphausiids in the upper estuary of the Gulf of St. Lawrence.** *J. Fish. Res. Bd. Can. 32(2): 310-314; 1975.* (14 references)

No significant variation was found in organochlorine residue levels between different stations in an area from the Saguenay River to the end of the Gaspe Peninsula in the Gulf of St. Lawrence for the euphausiids *Thysanoessa raschii, T. inermis*, and *Meganyctiphanes norvegica*. Significant differences were found between the concentration of organochlorine residues for *M. norvegica* and the *Thysanoessa* species, but there was no difference between the two *Thysanoessa* species. Significantly lower values for percent lipid were found in *T. inermis* and *T. raschii* between June 1973 and May 1974 samples, but the total amount of organochlorine residues did not change. This suggests that these species do not lose *p,p'*-DDT when they metabolize lipids for energy or the production of gonad material. (Author abstract by permission)

75-1280. Sekiguchi, F.; Morita, H.; Akimoto, T.; Oshima, Y. (Res. Lab. Daiichi Pharm. Co. Ltd., Tokyo, Japan). **[Organochlorine pesticide residues in laboratory animal foods.]** *Jikken Dobutsu (Lab. Anim.) 24(1): 13-18; 1975.* (19 references) (Japanese)

Total levels of organochlorine pesticide residues in diet for laboratory rats and mice, as determined by gas chromatography of acetone *n*-hexane extracts were 0.243 ppm in June 1970 and 0.044 ppm in June 1973. Levels of α-BHC varied widely, from 0.081-0.141 ppm in June 1970 to 0.009 ppm in June 1971, while levels of β- and γ-BHC showed little change. The level of *p,p'*-DDT was much lower in 1973 than in 1970-72. Throughout the survey (1970-73), aldrin and endrin were detected only slightly if at all. The diets for dogs, rabbits, monkeys, and chicks showed similar results. Total organochlorine pesticide residues in wheat bran and fish meal were higher, 0.317 and 0.103 ppm respectively, than in other ingredients of the animal diet, which contained 0.004-0.027 ppm.

75-1281. Lyubenko, P. Kh.; Stefanskiy, K. S.; Rozenfel'd, A. A. (All-Union Sci. Res. Inst. Hyg. Toxicol. Pesticides, Polymers, and Plastic Materials, Kiev, USSR). **Reglamenty primeneniya polikhlorkamfena v sel'skom khozyaystve i soderzhaniye yego v pochve i rasteniyakh. [Procedure in using polychlorocamphene in agriculture and its content in soil and plants.]** *Khim. Sel. Khoz. 11(12): 28-29; 1973.* (Russian)

A complex residue-dynamic and toxicological study on toxaphene and recommendations for its use in agriculture are described. The rate of degradation in soil was determined to be 50% in 12 months. Boiling for 6 hrs resulted in the degradation of 98% in water. Crops of toxaphene-treated potato, beet, carrot, cabbage, and parsley, containing 0.2 mg toxaphene or more per kg, were unacceptable organoleptically. Most of the residue was concentrated in the skin of the produce. Peeling and culinary preparation reduced the residue level to about one sixth of the original. The residue levels were highest in carrots, beets, parsley, and tomatoes. Toxicological tests revealed LD50 values of 45 mg/kg in mice and 240 mg/kg in rats. Toxaphene affected mainly the central nervous system and the spinal cord. Of all rats fed 0.22-2.2 mg/kg doses of toxaphene for 6 months, 26-43% died, and the surviving animals showed a decrease of the sensitivity threshold to electric pulses. The cumulation coefficient was determined to be 2.4 and 3.0 for 2.2 and 0.9 mg/kg doses. Changes in liver

functions, increased hippuric acid level in the urine, prolongation of hexobarbital-induced sleep, and changes in the succinate dehydrogenase level were observed in rats fed 4.8 and 2.4 mg/kg doses for 12 months. It is recommended that the toxaphene expenditure not exceed 1.5-2 kg/ha for perennial herbs. The waiting period before harvest should be 2.5 months in both cases. Toxaphene-contaminated soil should not be used for growing carrots, parsley, sugar beet, tomatoes, or medicinal herbs. The maximum allowable concentration of toxaphene in potaoto and sugar beet as well as in soil is 0.1 and 0.5 mg/kg, respectively.

75-1282. Klisenko, M. A.; Pis'mennaya, M. V. (All-Union Sci. Res. Inst. Hyg. Toxicol. Pesticides, Polymers, and Plastic Materials, Kiev, USSR). **Vliyaniye UF-sveta na stabil'nost' dialkilditiofosfororganicheskikh pestitsidov. [Effect of ultraviolet light on the stability of dialkyldithioorganophosphorus pesticides.]** *Khim. Sel. Khoz. 11(12): 916-918; 1973.* (5 references) (Russian)

The stability of the the dialkyldithioorganophosphorus pesticides phencapton, cidial (phorate), phosalone, phthalophos (phosmet), and menazon under UV light was studied in a wavelength range of 250-400 nm. The photodegradation of these compounds can be described by a first-order kinetic reaction. The half-life amounted to 12 min for phencapton, 11 min for cidial, 70 min for phthalophos, 86 min for phosalone, and 630 min for menazon. The transformation of the dialkyldithiophosphates studied under UV light is most probably due to the oxidation of the phosphorus-sulfur bond.

75-1283. Kuwahara, Y. (Sch. Pub. Health, Fac. Med., Kurume Univ., Kurume, Fukuoka, Japan). **[An epidemiological study on the health effects of the low-level long-term intake of organochlorine pesticides.]** *Kurume Igakkai Zasshi (J. Kurume Med. Assoc.) 37(11): 639-653; 1974.* (30 references) (Japanese)

β-BHC, *p,p'*-DDE, and *p,p'*-DDT contents in plasma of inhibitants in Chikugo area, Fukuoka Prefecture were determined and their occupational and local difference were epidemiologically investigated. The highest β-BHC residue values were found in a group of employees of a pesticides formulating factory (average: 82.04 ppb, *N*: 44), followed by medical school students (average: 81.0 ppb, *N*: 14). The β-BHC value in female farmers was significantly lower (average: 39.98 ppb, *N*: 385) than that in women in cities. On the contrary, the residual values of *p,p'*-DDE and *p,p'*-DDT in female farmers were the highest (average: 19.61 and 14.16 ppb) and showed significant local differences between 3 areas, possible due to the difference in agricultural procedures, as was also supported by the residual BHC/DDT ratios (10/15.99, 10/8.04, and 10/3.56). Although the value of β-BHC in female farmers showed a slight peak at ages of 50-55, that of *p,p'*-DDT plus *p,p'*-DDE showed a difference: higher in people older than 35 and lower in people younger than 35.

75-1284. Yoshida, K.; Nishikawa, M.; Matsumara, T. (Dep. Hyg. Chem., Nagasaki Prefect. Inst. Pub. Health Contr. Environ. Pollut., Nagasaki, Japan). **[Results of a survey on pesticide residues in foods. Part 4. Results in 1973.]** *Nagasaki-Ken Eisei Kogai Kenkyusho-Ho (Rep. Nagasaki Prefect. Inst. Pub. Health Contr. Environ. Pollut.) 13: 44-45; 1974.* (Japanese)

When pesticide residues were examined in 91 specimens of vegetables and fruit, only one specimen of potato contained dieldrin at 0.015 ppm more than the standard. As, Pb, HCB, and dieldrin were detected in potatoes, Chinese cabbage, spinach, pumpkin, grape, and strawberry, but at levels below the standards. In general, total BHC residues decreased in the order beef (0.092), pork (0.042), chicken meat (0.021), and eggs (0.012 ppm), and the first two types of meat showed a large reduction from the values for 1972. Total DDT was 0.093 ppm in pork, 0.027 ppm in eggs, 0.021 ppm in beef, and 0.018 ppm in chicken meat, showing a remarkable reduction from the values in 1972. The pesticide residues in market milk showed a reduction in total BHC (0.012-0.039 ppm), but did not show a reduction in DDT (0.012-0.002 ppm), or dieldrin (0.007-0.002 ppm). In mother's milk, the average residues of alpha-, beta-, gamma- and Δ-BHC, *p,p'*-DDT, *p,p'*-DDE, *p,p'*-DDD (TDE), and dieldrin were 0.001, 0.086, 0.002, N.D., 0.011, 0.052, 0.001 and 0.002. Pesticides residues (DDT and BHC) were lower in rural mother's milk than in milk of mothers who lived in the city.

75-1285. Zaborowska, W.; Witkowska, I.; Kozak, H. (Dep. Gen. Hyg. Acad. Med. Lublin, Poland). **Oznaczenie pozostalosci Pyrazonu w burakach. [Determination of pyrazon residue in red beets.]** *Rocz. Panstw. Zakl. Hig. 24(6): 735-740; 1973.* (10 references) (Polish)

Herbicide H-130 (containing 50% 1-phenyl-4-amino-5-chloropyridazone-6, PCA or pyrazon) was applied in amounts of 6.4 kg/ha (3.2 active substance) to experimental fields of red beets immediately after planting and 6 weeks later. Determinations of PCA residues in beets and leaves were made 5 times using thin layer chromatography (Dresher's method). After 5.5 weeks of growth and only one application of the herbicide 0 - 0.01 ppm were detected. After 8 weeks of growth and 2 applications of herbicide 0.04 – 0.150 ppm were found. After 11 weeks residues of PCA were 0.065 - 0.090 ppm, after 17 weeks (at time of harvest) 0 - 0.01 ppm, and after 24 weeks (6.5 weeks after harvest) no residues of PCA were present. Iso-PCA was detected in small amounts (< 0.0005 - 0.0005) in the second and third determinations.

75-1286. Kawahara, T.; Goto, S.; Fujimoto, Y.; Watanabe, T.; Maeda, H. (Agric. Chem. Inspect. Stn.,

Min. Agric. Forest., Tokyo, Japan). **[Variation of pesticide residues in fields. Part 1. Lead arsenate in apple orchards and fenitrothion in vineyards.]** *Noyaku Seisan Gijutsu (Pestic. Tech.) 27: 17-20; 1972.* (6 references) (Japanese)

Pesticide residues were determined in apple trees treated with lead arsenate and in grape vines treated with fenitrothion by estimating the variations of residue level in the fields. Variations of lead residues in apple trees were slight, and the analysis of variance revealed no significant differences in residues between upper and lower branches. Disappearance of lead residues from apple trees after application of lead arsenate proceeded at a moderate rate. Appreciable differences in arsenic residues were recognized among the parts of apple trees, with the lower branches containing twice as much arsenic as the upper ones. Arsenic disappeared from apple trees more rapidly than lead following application of lead arsenate. Great variation was found in the residues of fenitrothion in grape vines. The maximum determined was approximately five times as much as the minimum. Disappearance of fenitrothion in grapes after application was rapid. Differences in the residues attributable to type of applicator used were not apparent in these experiments. The crop sampling technique was singled out as one of the major factors determining pesticide residue variance.

75-1287. Annicchiarico Sebastiani, L.; Tarsitani, G.; D'Arca Simonetti, A. (Inst. Ig. "G. Sanarelli", Univ. Roma, Rome, Italy). **Comportamento di un erbicida nel suolo. Nota I. Movimento della prometrina nei terreni. [Behavior of a herbicide in soil. Part I. Migration of prometryne in the soil.]** *Nuovi Ann. Ig. Microbiol. 24(5): 339-349; 1973.* (11 references) (Italian)

The adsorption and passage of prometryne percolated through a column containing successively sand, a mixture of sand and clay, and native soil was studied to determine the penetration capacity of prometryne under the condition of saturation. The adsorption was highest in native soil, at 25-28% of the total input, followed by the sand-clay mixture, and finally by sand at 15-16%. The speed of the passage of prometryne through the successive layers decreased progressively. All soils tested permitted nearly quantitative recovery of prometryne at rates varying from 75 to 90% at the deepest point of 90-120 cm.

74-1288. Bronisz, H.; Ochynski, J. (Dep. Chem. Toxicol., Acad. Med., Lublin, Poland). **Zawartosc DDT i DDE w mleku kobiet wojewodztwa Lubelskiego. [DDT and DDE content in the milk of women in the province of Lublin.]** *Pediat. Pol. 48(4): 445-451; 1973.* (22 references) (Polish)

DDT and DDE contents were determined for the years 1968 and 1970 in 45 samples of milk from women in the Province of Lublin. Some of the women had no history of contact with pesticides while others used them in farming. In all cases the presence of DDT and DDE was discovered. In 1968 the overall mean value for both compounds was 0.50 mg/l (range 0.15-1.73 mg/l) while in 1970 this value was 0.36 mg/l (range 0.15-0.67). The difference in DDT content in the milk of women with or without contact with pesticides was considerable in 1968, when the amount of DDT in the milk of women without exposure to it was 0.43 mg/l, and in those who were exposed it was 0.61 mg/l. In 1970 this difference was insignificant: 0.35 and 0.38 mg/l. The results for the content of DDT in fat indicate that they approximately amount to the content of DDT in adipose tissue in children and exceed by 6–8 times the acceptable tolerance of DDT in cow's milk. The significance of DDT in milk is discussed.

75-1289. Bronisz, H.; Ochynski, J. (Dep. Chem. Toxicol., Sadowej Acad. Med., Lublin, Poland). **Zawartosc DDT i DDE w thance thuszczowej drieci wojewodztwa Lubelskiego. [DDT and DDE content in adipose tissue in children in the province of Lublin.]** *Pediat. Pol. 48(4): 453-458; 1973.* (17 references) (Polish)

DDT and DDE content were determined in 105 samples of adipose tissue from children aged up to 12 years in the Province of Lublin, over the years 1966 to 1970. Tissue was obtained on the first day after death, stored in 4% formalin, and tested by the colorimetric method of Schechter-Haller in the modification of Mattson. In 104 cases the presence of both compounds was discovered. In samples from the year 1966 the mean value of total DDT was 11.0 mg/kg, calculated for fat; in 1967, 7.5 mg/kg; in 1968, 8.8 mg/kg; in 1969, 9.5 mg/kg; and in 1970, 7.6 mg/kg. DDE accounted for about 62% of the sum of both substances. In 22 samples taken in 1970 from children aged up to 2 years, differences in the concentration of the insecticide were found to depend on age. The fat of children aged from 3 months to 2 years contained less DDT than that from babies aged up to 3 months. This is explained by the fact that the babies were breast fed and the milk of mothers contained high levels of DDT. Comparing results over the individual years a decrease of DDT content in fatty tissue is observed, similar to that observed in adults. This is due to the gradual limitation of the use of DDT in Poland in recent years. Values, however, are still high in relation to other countries.

75-1290. Kontek, M.; Paradowski, S.; Pietraszek, Z.; Kubacki, S. J.; Cieslicki, J.; Sniezynski, C. (Cardiol. Clin., Acad. Med., Poznan, Poland). **Zawartosc weglowodorow chlorowanych w tkance tluszczowej podskornej u osob z chorobami przewodu pokarmowego. [Chlorinated hydrocarbons in adipose tissue of persons with gastrointestinal diseases.]** *Pol. Tyg. Lek. 28: 860-862; 1973.* (13 references) (Polish)

Concentrations of DDT, DDE, DDD (TDE), and

BHC were determined in 15 samples of adipose tissue obtained from patients operated upon for gastrointestinal diseases. DDT (mean 5.72 ppm), DDE (mean 7.62 ppm), and BHC (mean 0.15 ppm) were found present in all samples. However, a minimal quantity of DDD (mean 0.004 ppm) was present in only 14 samples. The mean total concentration of DDT + DDE + DDD was 14.31 ppm.

75-1291. Lara, W. H.; Barreto, H. H. C. (Sec. Aguas, Inst. Adolfo Lutz, Sao Paulo, Brazil). **Residuos de pesticidas clorados em aguas. [Chlorinated pesticide residues in water.]** *Rev. Inst. Adolfo Lutz 32: 69-74; 1972.* (17 references) (Portuguese)

Chlorinated pesticide residues were determined by gas liquid chromatography with electron capture detection in 59 water samples received for chemical examination of their potability. Results indicate the presence of BHC residues (alpha, beta, and gamma isomers) in all samples examined. DDT was found in only 3 samples and traces of aldrin in one. The levels found were mostly in the 1-5 ppm range, indicating that contamination by these pesticides in waters of Sao Paulo, Brazil is presently not very great.

75-1292. Lara, W. H.; Barretto, H. H. C. (Sec. Adit. Pestic. Residuais, Inst. Adolfo Lutz, Sao Paulo, Brazil). **Residuos de pesticidas clorados em alimentos. [Chlorinated pesticide residues in foods.]** *Rev. Inst. Adolfo Lutz 32: 89-94; 1972.* (12 references) (Portuguese)

Samples of foods sold in Sao Paulo, Brazil were examined for organochlorine pesticide residues by GC-ECD. DDT-derivative residues were minimal; only traces were found in cereals and vegetables, and 0.2-0.2 ppm in various types of beans. Total BHC levels were 0.002 ppm in water, 0.009 ppm in rice, 0.035 ppm in beans, 0.038 ppm in manioc flour, 0.024 ppm in vegetables, 0.253 ppm in edible oils, 0.025 ppm in eggs, 0.032 ppm in milk, 0.390 ppm in meat, and 1.579 ppm in cheese. These results as well as those from previous surveys permitted the calculation of the daily intake of BHC by the population according to an estimated diet as 0.0004 mg/kg body weight/day. More than half of the daily intąke was accounted for by foods of animal origin.

75-1293. Beland, F. A. (Montana State Univ., Bozeman, MT 59715). **The use of molecular orbital calculations and electrochemistry to predict the reduction pathways of organochlorine compounds.** *Diss. Abstr. Int. 35(9): 4385B; 1975.*

The CNDO/2 molecular orbital method was used to investigate the electrochemical reduction of organochlorine compounds of environmental interest. As the degree of chlorination increased in chlorinated benzenes and biphenyls, the LUMO-σ and LUMO-π both decreased in energy. The HOMO of the radical anions for each of these species was always a σ orbital. The location of highest electron density in the LUMO-σ for all of the chlorobenzenes, DDT, lindane, and heptachlor predicted which chlorine was lost during electrochemical reduction. The electron density distribution in higher unoccupied σ-orbitals of DDT and heptachlor predicted the order of carbon-chlorine bond scission in succeeding reductions. Electrochemical reduction pathways correctly predicted the observed anaerobic degradation pathways for DDT, DTE, lindane, and hexachlorobenzene. 2,3,4,5,6-Pentachlorobiphenyl and decachlorobiphenyl were resistant to anaerobic reduction. The first electrochemical reduction product of decachlorobiphenyl did undergo anaerobic reduction. During anaerobic degradation heptachlor lost the allylic chlorine first as opposed to the "anti" methylene bridge chlorine observed electrochemically. These results indicate that if a compound has an E_2d more cathodic than -1.75 V (vs. SCE) in a DMSO-TEABr solvent system, it will not reduce in an anaerobic environment. Compounds with an E_2d more anodic than this value may be reduced in the environment. Whether they do or not seems to depend on their actual structure which indicates that these compounds may have to fit into some type of an "active site." (Author abstract by permission. Copies of the thesis are available from University Microfilms, Order No. 75-5318.)

75-1294. Cwiertniewska, E. (Dept. Anal. Chem., Nat. Inst. Hyg., Warsaw, Poland). **Pozostalosci kaptanu i metoksychloru w niektorych owocach. [Residues of captan and methoxychlor in certain fruits.]** *Rocz. Panstw. Zakl. Hig. 24(3): 309-314; 1973.* (21 references) (Polish)

A method of thin-layer chromatography is described such that it is possible to simultaneously detect residues of captan in concentrations of 1 mg/kg and methoxychlor at 0.4 mg/kg in pears or apples with almost 100% recovery of both compounds. These concentrations are much lower than the proposed maximum residues which are 6 mg/kg for captan and 10 mg/kg for methoxychlor. No pesticide residues were detectable by this method in any of 40 fruit samples examined.

75-1295. Obuchowska, I. (Dep. Communal Hyg., Nat. Inst. Hyg., Warsaw, Poland). **Badania nad zachowaniem sie tritoxu-30 w glebie. [A study of Tritox-30 in soil.]** *Rocz. Panstw. Zakl. Hig. 23(2): 147-154; 1972.* (19 references) (Polish)

The vertical movements and displacement of the preparation "Tritox liq. 30", containing the insecticides DDT, methoxychlor (DMDT), and gamma-BHC (lindane) as active ingredients are reported. A field experiment was carried out using a plot contaminated by this preparation in 1969. The plot and the adjacent area were submerged under water for 2-2.5 months during

the period of observation in 1971. By means of thin-layer chromatography, it was found that DDT and methoxychlor moved into the soil down to 20 cm with the higher concentrations (8 mg/kg DDT and 6 mg/kg DMDT) in the upper 10 cm layer. No lindane could be detected. During the first several months of the study, the levels of DDT and methoxychlor were constant. The apparent degradation of both compounds took place only after flooding (62.5% of DDT and 50% of DMDT being detected following the event). The horizontal displacement has been found to reach limits of 100-400 cm beyond the experimental plot, where only trace amounts of both compounds could be detected.

75-1296. Cwiertniewska, B.; Dominska, J.; Kowianska, E. (Dep. Anal. Chem., Nat. Inst. Hyg., Warsaw, Poland). **Badanie mleka surowego i sproszkowanego na zawartosc ogolnego DDT i HCH. [An inquiry into total DDT and BHC residues in raw and powdered milk.]** *Rocz. Panstw. Zakl. Hig. 23(3): 271-278; 1972.* (2 references) (Polish)

Over the period of May 1970 to April 1971 the residues of total DDT and BHC in raw and powdered milk were estimated. In 10 percent of the samples of raw milk the level of pesticides amounted to 1.25 ppm DDT and 0.1 ppm BHC on a fat basis and thus was higher than the permissible values recommended by FAO/WHO. A distinct rise in the levels of both constituents in milk was observed in July and August as well as from November to February inclusive. A 19-100 percent diminution of the compounds under study was found in all samples of powdered milk in comparison to raw milk except for those manufactured in June, November, and January.

75-1297. Inuyama, Y.; Takashita, T. (Shimane Prefect. Inst. Pub. Health Contr. Environ. Pollut., Matsue, Shimane, Japan). **[Survey of pesticide residues and PCB in mother's milk and human adipose tissue.]** *Shimane-Ken Eisei Kogai Kenkyusho Nenpo (Annu. Rep. Shimane Prefect. Inst. Pub. Health Environ. Pollut.) 15: 37-39; 1973.* (Japanese)

Ten specimens of milk from human mothers (between 1 and 4 months after delivery), 20 specimens of human adipose tissue removed at surgery, 3 specimens of adipose tissue from birds (2 crows and 1 heron), and 1 specimen of sea gull egg were analyzed for α-, β-, and γ-BHC (lindane), DDT, DDD (TDE), DDE, and polychlorocyclodienes. The average amounts in mother's milk were 0.003, 0.0851, 0.0013, 0.0213, ND, 0.0820, 0.0051 (dieldrin), and ND (aldrin and endrin), showing a tendency for β-BHC and dieldrin to be higher in cities than in rural areas and an increasing trend for DDT. The pesticide residues in adipose tissue from elderly humans showed average total BHC of 3.4317, total DDT of 6.2825, and dieldrin of 0.1594 ppm, thus a decreasing trend for total BHC and dieldrin and increasing trend for total DDT. The residues of total BHC, total DDT, and dieldrin in adipose tissue of crows were on the average 10.9167, 25.1676, and 4.1979 ppm, showing a high level of pollution and suggesting the suitability of crows as a biological indicator.

75-1298. Inuyama, Y. (Shimane Prefect. Inst. Pub. Health and Control of Environ. Pollut., Matsue, Shimane, Japan). **[Results of survey on pesticide residues in foods.]** *Shimane-Ken Eisei Kogai Kenkyusho Nenpo (Annu. Rep. Shimane Prefect. Inst. Pub. Health Environ. Pollut.) 15: 39-41; 1973.* (Japanese)

Pesticide residues in 75 specimens of vegetables and fruits, 24 specimens of cow's milk, 20 specimens of pork, and 8 specimens of internal organs of swine were determined in 1973. Of officially designated pesticides, 0.008 ppm γ-BHC and 0.04 ppm DDD (TDE) were each found in only one specimen of peel of summer orange, and 0.007-0.01 ppm dieldrin was found in 3 specimens of cucumber. In cow's milk, residues of α-, β-, and γ-BHC, DDT, DDE, DDD, and dieldrin were max. 0.02, 0.06, 0.001, 0.001, 0.002, 0.001, and 0.001, and average 0.001, 0.0027, 0.0002, 0.0007, 0.0012, 0.0003, and 0.0007 ppm, respectively, showing a tendency of β-BHC and a less remarkable tendency of DDT and dieldrin to decrease. In pork, average residues were, in the above order, 0.0115, 0.0108, 0.0021, 0.3512, 0.0135, 0.1707, and 0.003 ppm, showing a decreasing trend for β-BHC and dieldrin, and on the contrary an increasing trend for DDT. Examination of internal organs showed the highest concentrations of β-BHC, dieldrin, and DDT to be in the liver. Residues found were highest in the case of DDT, followed by β-BHC, and then by dieldrin.

75-1299. Craig, R. B.; Rudd, R. L. (Dep. Zool., Univ. California, Berkeley, CA). **The ecosystem approach to toxic chemicals in the biosphere.** *IN: Survival in Toxic Environments, M. A. Q. Khan and J. P. Bederka, Jr., eds., Academic Press, New York, 1974, p. 1-24.* (62 references)

A mathematical model of the kinetics of chlorinated hydrocarbon residues in aquatic ecosystems was developed. It rests upon physical, chemical, biological, and ecological data using Clear Lake, California, as an example. Computer analyses of the DDT-R (DDT plus DDD (TDE) and DDE) load throughout the lacustrine ecosystem are presented together with descriptive and predictive models of the pesticide residue values found in fishes and one fish-eating bird, *Aechmophorus occidentalis*. Death of adults followed by reproductive failure in the remaining population is attributed to chlorinated hydrocarbon accumulation in tissues. Projections indicate no relief from partial reproductive inhibition for several years. The ecokinetic analysis of chemical residues was extended to a terrestrial ecosystem whose complexities are reduced by intensive agriculture. Material flow and pesticide residue distribution are analyzed for the simplified agricultural ecosystem in the San Joaquin Valley, Calif.

75-1300. Ohlendorf, H. M.; Klaas, E. E.; Kaiser, T. E. (U.S. Bureau of Sport Fisheries and Wildlife, Patuxent Wildlife Res. Cent., Patuxent, MD). **Environmental pollution in relation to estuarine birds.** *IN: Survival in Toxic Environments, M. A. Q. Khan and J. P. Bederka, Jr., eds., Academic Press, New York, 1974, p. 53-81.* (39 references)

The adverse effects of organochlorine pesticides in birds were investigated by examining eggs of anhingas (*Anhingidae*), herons (*Ardeidae*), and ibises (*Threskiornithidae*) collected during the 1972 nesting season at coastal and inland localities from Florida to New Jersey. Eggshell thinning was not detected among those species for which adequate samples were available for pre-1946 and post-1945 eras. Measurable residues of DDE occurred in all 209 eggs. The highest mean value (4 ppm, wet weight) was found in great egrets from New Jersey. Among the coastal localities, levels of DDE as well as total DDT progressively declined toward the south. Polychlorinated biphenyls occurred second most frequently and also reached their highest mean level (4.2 ppm) in the great egret eggs from New Jersey. Higher levels of polychlorinated biphenyls were found in eggs from eastern Florida (anhinga, 2 ppm). Great egrets carry higher DDT residues; glossy ibises, snowy egrets, and little blue herons have residues at intermediate levels, and other species have residues at lower levels.

75-1301. Khan, M. A. Q.; Khan, H. M.; Sutherland, D. J. (Univ. of Illinois at Chicago Circle, Chicago, IL). **Ecological and health effects of the photolysis of insecticides.** *IN: Survival in Toxic Environments, M. A. Q. Khan and J. P. Bederka, Jr., eds., Academic Press, New York, 1974, p. 333-355.* (47 references)

The ecological and toxicological effects of the photolysis of insecticides are reviewed. Photolysis occurs on the surface or in solution either by direct electronic excitation following light absorption or through sensitizers. Studies were made of the photolysis of DDT and its analogs DDE and methoxychlor. On exposure to ultraviolet light (2537 A) aldrin undergoes epoxidation to dieldrin, dechlorination to pentachloroaldrin, and cage isomerization to photoaldrin. Sulfur-containing compounds which are readily photooxidized include demeton, parathion, abate (temophos), and rotenone. Some of the safest and most useful compounds, such as methoxychlor, have limited use due to their photodecomposition. The most significant aspect of photolysis of insecticides is that some of the most commonly used ones, aldrin, dieldrin, and parathion, are converted to more toxic products. The LC50 values of various cyclodienes and their photoisomers to houseflies and aquatic animals are tabulated.

see also 75-1313 75-1327 75-1329 75-1340

75-1302. Palva, H. L. A.; Koivisto, O.; Palva, I. P. (Dep. Med., Univ. Oulu, Oulu, Finland). **Aplastic anaemia after exposure to a weed killer, 2-methyl-4-chlorophenoxyacetic acid.** *Acta Haematol. 53(2): 105-108; 1975.* (5 references)

A 64-yr-old farmer developed aplastic anemia after exposure to 2-methyl-4-chlorophenoxyacetic acid (MCPA) in a weed killer. The man had a history of coronary heart disease extending back several years, and regularly took digoxin and nitroglycerin tablets. During the last 5 yr he had sprayed chemicals on weeds on his farm. The previous summer he employed an old manual spraying device with the container on his back. The container leaked so that his clothes became wet with the spraying solution. Two weeks after the last spraying he detected spontaneous hematomata on his body and felt lethargic. He waited 2 months before consulting a doctor. Muscular weakness, hemorrhagic gastritis and slight signs of liver damage were noted. Serum iron was 32 μmol/l, TIBC 40 μmol/l, B_{12} 700 pmol/l, and folate 10.5 mmol/l. All symptoms were consistent with toxic effects of chlorophenoxyacetic acids recorded in animal experiments. Transfusions of packed red cells were given. He also received prednisolone (80 mg/day, p.o.) for 10 days, then methenolone (100 mg/day). Five months later he was free of symptoms. This case demonstrates the necessity of observing strict safety rules in the use of such insecticides.

75-1303. Ott, M. G.; Holder, B. B.; Gordon, H. L. (Dow Chem. Co., Corp. Med. Dep., Midland, MI 48640). **Respiratory cancer and occupational exposure to arsenicals.** *Arch. Environ. Health 29: 250-255; 1974.* (12 references)

The proportionate mortality experience of 173 decedents exposed primarily to lead arsenate and calcium arsenate was compared with that of 1,809 decedents not exposed to those compounds. A significant increase in respiratory cancer was found among the exposed employees. The relationship between cumulative arsenic exposure and the ratio of observed to expected respiratory malignancy deaths was estimated by means of a least squares approach. The predicted ratio was 7:1 for individuals exposed for a period of more than eight years to compounds that contained an equivalent level of 1 mg/m^2 arsenic. In the more heavily exposed individuals, an excess of respiratory cancer was observed 35+ years after the initial exposure. Observations based on the proportionate study were supported by an analysis of the same target population, employing the prospective study in retrospect approach. (Author abstract by permission)

75-1304. Gehlbach, S. H.; Williams, W. A. (North Carolina Div. Health Serv., Raleigh, NC 27602). **Pesticide containers.** *Arch. Environ. Health 30: 49-50; 1975.* (3 references)

Nine case histories of container-related pesticide poisonings are presented. Inadequate provisions for container disposal were responsible for three poisoning cases involving children playing with "empty" pesticide containers. Inappropriate container design was blamed for one fatality in which a pesticide solution was mistaken for drinking water, and three more cases, one of them fatal, occurred due to pesticide release from fragile glass and fabric containers. Two toddlers playing with a non-safety package of insect spray were also poisoned. One poisoning case involved a carbamate, oxamyl; the others were all organophosphates, mostly parathion. Diagnosis was facilitated by the prominent nicotinic and muscarinic symptoms, and vigorous treatment with atropine and oximes was successful if started in time. Imposition of a cash deposit on pesticide containers might encourage their return and reuse. The use of containers which resemble food or beverage containers should be prohibited.

75-1305. Futenma, M. (Dep. Ophthalmol., Juntendo Univ. Sch. Med., Bunkyo-ku, Tokyo, Japan). **[A screening of the visual field on 13,357 school children.]** *Nippon Ganka Gakkai Zasshi (J. Jpn. Ophthalmol. Soc.) 77(8): 719-730; 1973.* (14 references) (Japanese)

Because of the recent reports of visual field abnormalities supposedly caused by organophosphate pesticides or formalin exposure, visual field screening was incorporated into the school health program. In the area of the study, orange growing is a major industry, and the children are more heavily exposed to insecticides than urban children. Examinations were performed with a new rapid portable perimeter with moving flash lights on a vertical and horizontal arc, using the multiple pattern method. Of 13,351 school children examined, 32 had visual field abnormalities. These 32 children were referred to a hospital for repeated visual field examination with a Zeiss spherical perimeter. Twelve of the children were abnormal in this test. Two of these showed organic abnormalities: one had retinitis pigmentosa, and the other had homonymous hemianopsia due to previous meningitis. Three children declined follow-up. Visual field abnormalities were diagnosed as psychological in the case of five children in whom the abnormalities disappeared after elimination of psychological disturbances. No definite reasons for the visual field abnormalities were found in the remaining two children, one of whom had a history of carbon monoxide poisoning at the age of nine months while the other had had asphyxia neonatorum.

75-1306. Oliver, R. M. (Civil Service Med. Service, Whitehall, London, England). **Toxic effects of 2,3,7,8-tetrachlorodibenzo-1,4-dioxin in laboratory workers.** *Br. J. Ind. Med. 32(1): 49-53; 1975.* (18 references)

Toxic effects in 3 young male scientists (aged 28-38 yr) who had transient minimal exposure to

2,3,7,8-tetrachlorodibenzo-1,4-dioxin are described. Two of the men suffered from typical chloracne. Delayed symptoms about 2 yr after initial exposure occurred in 2 of the scientists. These symptoms included personality changes, other neurological disturbances, and hirsutism. All had serum cholesterol in excess of 7.8 mmol/l, but no other biochemical disturbances, and no porphyrinuria nor liver damage was demonstrated. The history of these 3 patients emphasizes that dioxin must be regarded as highly toxic and that even those working with laboratory quantities with normal precautions are at a significant risk. Intoxication may occur in the absence of abnormal liver function tests on elevated urinary porphyrins. Such profound long-term effects as those seen here suggest that the dioxin molecule is highly active when absorbed into the body in minute quantities.

75-1307. Kay, K. (Environ. Sci. Lab., Mount Sinai Sch. Med., City Univ. New York, New York, NY 10029). **Occupational cancer risks for pesticide workers.** *Environ. Res. 7(2): 243-271; 1974.* (176 references)

There has been concern in recent years about the carcinogenic potential of pesticidal residues in food. Since pesticide workers receive much higher exposure to such chemicals at the work level, an assessment of the range of materials with carcinogenic potential has been made by a review of the literature. One hundred and seventy-five papers bearing on the subject were found. Their review has disclosed that many pesticidal chemicals possess at least low levels of carcinogenic activity for laboratory species. The review has demonstrated the major effect of spontaneous tumor incidence when chemicals of low cancer producing potential are being evaluated. It has also been established that some carriers and vehicles of pesticidal chemicals are carcinogenic or co-carcinogenic in man. There is some evidence of synergistic action between ingredients of particular preparations but no indication of additive carcinogenic potential among the various pesticides studied. (Author abstract by permission of Academic Press)

75-1308. Sano, I. (Joint Study Unit on Toxic Liver Injury, Natl. Med. Cent. Hosp., Tokyo, Japan). [Toxic liver injury.] *Iryo (Med. Treat.) 29(2): 155-165; 1975.* (15 references) (Japanese)

A survey was made on clinical-pathological findings in 158 cases of toxic liver injury in 27 national hospitals and results were reported. The toxic substances were 35 antibiotics including chloramphenicol and novobiocin, both also used as agricultural fungicides; 11 sulfa drugs; and 112 other drugs. It was noted that 2 cases were due to organophosphorus pesticides designated by law as toxic substances. Of 158 cases, 65 were examined histopathologically and findings on the progression of internal symptoms were described. The cases due to organophosphorus pesticides were not described further.

75-1309. Beasom, S. L. (Dep. Wildl. Fish. Sci., Texas Agric. Exp. Stn., College Station, TX 77843). **Selectivity of predator control techniques in South Texas.** *J. Wildl. Manage. 38(4): 837-844; 1974.* (10 references)

During a comprehensive study to determine the role of predation in reducing the productivity of the wild turkey (*Meleagris gallopavo intermedia*), data was gathered on the selectivity and efficiency of M-44's, hunting, strychnine baits, and steel traps for taking certain predators. Selectivity is that property of taking only target species which, for the present study, were coyotes (*Canis latrans*), bobcats (*Lynx refus*), and other small predatory furbearers. The M-44 and hunting were the most selective methods, each taking only target species. Strychnine baits and steel traps took a variety of animals including game animals, raptors, rodents, song birds, and reptiles. The M-44 was probably the most efficient tool for taking coyotes while bobcats were taken more efficiently with steel traps. Nontarget-species in one area may be target species in another area so care should be exercised in generalizing about selectivity of predator control techniques. (Author abstract by permission)

75-1310. Div. Protect. Living Environ. (Ehime Prefect. Off., Ehime, Japan). [Environmental pollution by agricultural chemicals (pesticides): cases of pesticide poisoning.] *IN: Ehime-Ken No Kogai (Environmental Pollution in Ehime Prefecture), Ehime Prefect. Office, 1974, p. 205-207.* (Japanese)

All cases of intoxication by agricultural pesticides reported between 1961 and 1973 were listed and classified as accidental cases (resulting during application or from misuse) and intentional cases (attempted suicide and murder), fatal and nonfatal. The total numbers of accidental poisonings and fatalities respectively were 11 and 34 in 1969, 23 and 21 in 1970, 9 and 21 in 1971, 38 and 24 in 1972, and 26 and 15 in 1973. Most of the fatalities were due to suicide and murder, accounting for 11 in 1969, 20 in 1970, 18 in 1971, 21 in 1972, and 15 in 1973. The poisonings developed during pesticide application (number of deaths: 2 in 1961, 1 in 1970, and 1 in 1972) might have been due to the lowering of risk-consciousness after long experience in application, or to long-term application under unhealthy conditions. Because there is no requirement for official reporting of such intoxication cases, there may be considerable differences between the above-mentioned numbers of cases and the actual numbers.

75-1311. Coppola, A.; Totaro, S.; Di Blasi, S.; Morici, G.; Panno, G. (Inst. Med. Lab., Univ. Palermo, Palermo, Italy). **Sulla patologia da brumuro dimetile. Rilievi su un gruppo di soggetti esposti e considerazioni di ordine preventivo.** [Methyl bromide pathology. Findings in an exposed group and safety considerations.] *Folia Med. (Naples) 54(12): 311-325; 1971.* (32 references) (Italian)

A review is presented of the physical and chemical properties, uses (fumigant, herbicide, nematicide, greenhouse and soil disinfectant and insecticide, insecticide for foodstuffs, wood, hides, etc.), and symptoms of methyl bromide poisoning. Results are reported from the pre-employment examinations of 78 subjects (mainly student summer-vacation workers aged 23 years on the average) employed in insect eradication in glasshouses, and the periodic examination of 41 of these subjects. Findings (pruriginous erythema of hands, palmar desquamation, persistent headache and nausea, vertigo) indicate the need for rigorous safety measures (informing workers of the hazards, hermetic sealing of containers, storage in a cool, dry place sheltered from heat, careful handling, prohibition of the use of grease to lubricate valves, general and local exhaust ventilation, personal protective equipment).

75-1312. D'Angelo, A. M.; Losito, L. (Inst. Hyg., Univ. Bari, Bari, Italy). **Gli avvelenamenti da pesticidi in Puglia dal 1958 al 1968. [Pesticide intoxication in the Puglia region from 1958 to 1968.]** *Ig. Mod. 65(1-2): 40-51; 1972.* (19 references) (Italian)

An epidemiological study was carried out on the frequency of the intoxication by pesticides in Puglia (Southern Italy) from 1958 to 1968. On the basis of data collected from the records of the Ospedale Consorziale in Bari over this period 178 cases were recorded (152 cases by parathion) and the mortality rate was 91.5%. The cases were classifed according to age, sex, and town of residence, as well as form (occupational, accidental, suicide, and homicide) and modes of poisoning. The highest number of poisonings was found among females, and among children under 10 years of age. Accidental poisoning accounted for 75.2%, followed by occupational, professional 14%, and intoxication due to suicide 10.1%. A total of 89 subjects (50%) were orally intoxicated, 78 (43.8%) were poisoned through the skin, and 11 (6.1%) through the respiratory tract.

75-1313. Yamada, A. (Sch. Pub. Health, Fac. Med., Kurume Univ., Kurume, Fukuoka, Japan). **[Effects of organochlorine pesticide residues on maternal and infant health.]** *Kurume Igakkai Zasshi (J. Kurume Med. Assoc.) 37(11): 654-681; 1974.* (46 references) (Japanese)

Blood specimens were collected from 130 pregnant women and from the corresponding umbilical cords after delivery, and residues of organochlorine pesticides were determined in the plasma. All 130 paired plasma specimens contained β-BHC, *p,p'*-DDT and *p,p'*-DDE; the levels of all three organochlorine pesticide residues were significantly higher in maternal plasma than in that of the umbilical cords. Higher plasma β-BHC levels (282 ppb) were frequently associated with premature births, abruptio placentae, incomplete uterine contraction, inadequate lactation, and growth retardation of the newborns. The β-BHC and total-DDT levels in maternal plasma were correlated with the umbilical cord plasma levels. Animal studies were performed, involving the determination of β-BHC in the plasma of maternal and new born Sprague-Dawley rats after administration of β-BHC to the mothers at 10 mg/kg. Clinically and experimentally, the placental penetration capacity of organochlorine pesticides was shown to be, in decreasing order, *p,p'*-DDT, β-BHC, and *p,p'*-DDE. It was concluded that organochlorine pesticides are transferred from mother to fetus via the placenta, and to newborns via the mother's milk.

75-1314. McCaughey, H. (Poison Information Cent., Royal Alexandra Hosp. Children, Camperdown, Australia). **Paraquat overdose.** *Med. J. Aust. 2(23): 855-856; 1974.* (10 references)

The lethal dose of paraquat has been the subject of much speculation. Absorption is known to be sporadic and low and thus ingested amounts must not be confused with injected amounts. The use of animal data to predict a lethal dose for man has not been particularly helpful. Few cases exist among human poisonings in which a good estimate of dose has been obtained. Various dose levels have been reported and the outcome differed from patient to patient. The total body of evidence suggests that the LD50 of paraquat in man is very roughly 30 mg/kg. It has been suggested that the granular form is less toxic than the aqueous. Perhaps the corrosive action of the aqueous solution aids intestinal absorption; perhaps the granules do not dissolve in the part of the gut where maximum absorption takes place; perhaps the magnesium sulfate present in the granular formation speeds transit time sufficiently to keep absorption down. It is suggested that a 20% granular formation be developed and tested.

75-1315. Simpson, G. R. (Div. Occup. Health Pollut. Control, Lidcombe, New South Wales, Australia). **Blood cholinesterase survey. Blood cholinesterase levels of persons exposed to organic phosphate pesticides in the agricultural field.** *Med. J. Aust. 2(24): 869-871; 1974.* (2 references)

A widespread blood cholinesterase survey was conducted during the 1974 spraying season to assess the overall exposure situation among agricultural workers in the field. The primary areas surveyed were Wee Waa and Trangie. Of 952 persons studied, 5 were seriously affected with blood cholinesterase levels below 40% of normal. A further 16 persons had blood cholinesterase levels below 60% of normal. In general, these persons were associated with aerial spraying operations in cotton fields. It was noted that the people were not using proper protective clothing during the handling of the toxic chemicals. Market gardeners also showed some group evidence of lowered cholinesterase levels. The pesticides used in cotton spraying included chlorcam-DDT, endrin-DDT, monocrotophos, and parathion. The

most popular pesticide used in orcharding was azinphos plus some light applications of methidothion, vamidothion, and leptophos. Tobacco growers used parathion, azinphos, methomyl, and aminocarb, plus some methyl demeton. Terracur and nemacur were used in soil treatment. Diazinon was the main chemical used for fly control in grazing. Fenthion-ethyl was also used. Tomato growers used terracur and nemacur, methyl demeton, parathion, methomyl and mevinphos. This data indicates that in the areas of market gardening and aerial field spraying, adequate protective measures are not being observed.

75-1316. Ouw, K. H.; Shandar, A. G. (Div. Occup. Health Radiat. Control, Health Commission, Lidcombe, New South Wales, Australia). **A health survey of Wee Waa residents during 1973 aerial spraying season.** *Med. J. Aust. 2(24): 871-873; 1974.* (13 references)

In April 1973 a health survey was conducted in the Wee Waa area to determine whether aerial pesticide spraying of area cotton fields had constituted a health hazard to Wee Waa residents. Aerial spraying was used to control Boll worms (*Heliothis punctigera* and *H. armigera*). During the 1971-72 spraying season many pesticide poisonings were recorded among occupationally exposed workers. During 1973, chlorinated hydrocarbons were used, and the chlorinated hydrocarbon levels in blood and whole blood cholinesterase activity were estimated. Of 76 volunteers examined in the Wee Waa and Narrabri areas, 47 had no history of pesticide exposure. The remaining 29 were exposed either occupationally or at home. Pesticides used in aerial spraying included DDT, endrin, chlorinated camphene (toxaphene), dichlorvos, mevinphos, parathion, and monocrotophos. Whole blood cholinesterase activity test results were normal in all but 4 cases. Three of these had occupational exposure; the other had no pesticide exposure. In the latter case, no symptoms were noted, but the cholinesterase activity was 47%. In the other 3 cases, in which cholinesterase activity ranged from 44 to 55%, symptoms included headache, nausea, abdominal pain, tiredness, and weakness. Only negligible amounts of pesticides were found in samples of water from roof tanks and from the town supply. The mean DDT levels in the exposed and non-exposed groups were 21.9 and 16.7 ppb, respectively. The mean hexachlorobenzene blood level in Wee Waa was 47 ppb, which is within the acceptable range. It is concluded that aerial spraying did not constitute a health hazard to the non-occupationally exposed population of the Wee Waa district.

75-1317. Davies, R. E. (Rural Division, ICI Australia Ltd., Melbourne, Victoria, Australia). **The paraquat puzzle.** *Med. J. Aust. 1(2): 45; 1975.* (1 reference)

Recent animal studies in the United Kingdom have indicated that ingested paraquat should be removed not only from the stomach but also from the remainder of the gastrointestinal tract. It is advised that the stomach be washed out with care because of possible esophageal damage, and, after washout, a large volume of 30% suspension of Fuller's earth be given together with an effective cathartic. This administration of Fuller's earth and cathartics should be repeated as frequently as is practicable, every 2 to 4 hr, for several days. Subsequent management plus information regarding local toxicity and the determination of paraquat poisoning are contained in a brochure entitled *The Treatment of Paraquat Poisoning: A Guide to Doctors*, which has been distributed to poisons centers and hospitals in all States of Australia.

75-1318. Faure, J.; Marka, C.; Faure, H.; Yacoub, M.; Cau, G. (Soc. Med. Leg. Criminol. de France, Paris, France). **Donnees histo-pathologiques et toxicologiques d'une intoxication mortelle par le paraquat. [Histopathological and toxicological symptoms of a fatal intoxication by paraquat.]** *Med. Leg. Dommage Corpor. 6(4): 417-419; 1973.* (21 references) (French)

Histopathological investigations on a 25-year-old man who died due to intentional ingestion of paraquat are reported. Congestion of the lungs, serous catarrhal alveolitis, interalveolar fibrosis, intrahepatic cholestasis, and epithelial cells with granular cytoplasm in the convoluted tubules of the kidney were determined on autopsy. Retention jaundice with anuria followed by major respiratory disturbances, diffuse pulmonary interstitial fibrosis, and toxic acute tubular necrosis in the kidney were found as characteristic symptoms of poisoning with paraquat. Both the retention jaundice and the lung fibrosis developed in a very short time, in a matter of a few days, while paraquat could not be detected in the blood 4 days after poisoning. Paraquat was detectable in the feces for several days.

75-1319. Conso, F.; Guillam, C.; Bescol-Liversac, J. (Soc. Med. Leg., Paris, France). **Intoxication par le dimethylpyridylium (paraquat). Commentaire des images observees en microscopie electronique. [Paraquat poisoning. Comments on electron microscopic observations.]** *Med. Leg. Dommage Corpor. 6(4): 420-422; 1973.* (1 reference) (French)

The action of paraquat on the human body is described on the basis of three acute poisonings, including one fatal case, due to the accidental ingestion of commercial paraquat solution. The clinical evolution of the poisoning occurs in three phases; in digestive phase with lesions of the mucosa; renal phase with predominant epithelial nephritis; and a pulmonary phase characterized by irreversible lesions and rapidly progressing diffuse sclerosis resulting in often fatal respiratory insufficiency. Paraquat, denaturating the lipid acids, exerts a toxic effect on certain sensitive cell membrane formations, especially in renal cells and pneumocytes

(especially type II pneumocytes), resulting in a deterioration of the respiratory mechanism on a cellular and tissular level in connection with a surfactant deficiency. The inflammatory reaction of the alveolar septa is a result of the epithelial alterations. The granulomatous transformation of the septal interstitium leads to a progressive and rapid sclerosis.

75-1320. Marcinkowski, T.; Manikowski, W. (Dep. Occupational Med., Acad. Med., Poznan, Poland). **Zejscie smiertelne w nastepstwie dolegliwosci, ktore wystapily po pracy przy zaprawie nasiennej T. [Fatal case of intoxication with "Seed Dressing T".]** *Med. Pracy 24(1): 91-95; 1973.* (2 references) (Polish)

A case is reported of a man, working at seed dressing with "Seed Dressing T" (containing about 50% tetramethylthiuram disulfide (thiram) on a mineral carrier) over a period of 10 hours, who developed dyspnea, general weakness, strong lacrimation, and respiratory tract irritation (of the larynx in particular) which resulted in aphonia. On the fourth day after exposure he died and histopathological findings at autopsy revealed lobular bronchopneumonia. The man was working under conditions contrary to rules of occupational safety and hygiene (no protective mask or gloves), and used a spade for mixing the grain with Seed Dressing T instead of a seed dressing machine.

75-1321. Kontek, M.; Pietraszek, Z.; Paradowski, S. (Cardiol. Clin., Inst. Internal Med., Acad. Med., Poznan, Poland). **Zachowanie sie aktywnosci cholinesteraz osoczowej i krwinkowej u pracownikow rolnych narazonychnna dzialanie insektycydow fosforoorganicznych. [Serum and erythrocyte cholinesterase activity in agricultural workers exposed to organophosphate and carbamate insecticides.]** *Med. Pracy. 24(2): 213-218; 1973.* (8 references) (Polish)

Twenty-four agricultural workers were exposed for 21-24 days to organophosphorus compounds such as Birlane (chlorfenvinphos), Intration (thiometon), and BI-58 (dimethoate) (Group I) and organophosphate and carbamate compounds such as BI-58 (dimethoate), Sapekron (chlorfenvinphos), and Unden (propoxur) (Group II). The activities of serum and erythrocyte cholinesterase were determined during the final days of exposure and over a 65-day period after cessation of exposure. Significant lowering of SChE activity was observed during the final days (in group I to 0.18 ΔpH/hr on the average, in group II to 0.36 ΔpH/hr) and relatively slight lowering of EChE activity (on the average to 0.54 ΔpH/hr and 0.57 ΔpH/hr in Group I and II, respectively). Recovery was slow. In group I at 65 days after exposure had been discontinued, SChE and EChE activity reached levels of 0.55 and 0.58, respectively. In group II, after 56 days, they equaled 0.54 and 0.57 ΔpH/hr, respectively.

75-1322. Wakasugi, C.; Fukui, M. (Sch. Legal Med., Fac. Med., Osaka Univ., Osaka, Japan). **[A case report of fatal intoxication by ingestion of calcium polysulfide.]** *Nippon Hoigaku Zasshi (Jpn. J. Leg. Med.) 28(2): 102-103; 1974.* (5 references) (Japanese)

Intoxication, possibly by ingestion of the fungicide calcium polysulfide, was reported in the suicide of a 22-year-old student. External findings were a yellowish white substance with strong sulfide odor adhering to the head, face, and hands; eroded conjunctiva; and postmortem hypostasis of dark blood. An empty 400 ml bottle labeled as containing calcium polysulfide was found beside the corpse. Findings at autopsy were: somewhat dark and slightly thick blood in the heart; conspicuous edema and extensive hemorrhage in the lungs; darkening of liver, pancreas, kidney, and brain (especially the cortex); eroded and moderately indurated mucosa of laryngopharynx, trachea, bronchioles, esophagus, stomach, and upper part of small intestine; yellowish white erosion and coagulation of the surface of liver and intestine; 100 ml of brown turbid stomach content with strong sulfide odor. Methemoglobin sulfide was detected in blood in the heart. Vacuolar denaturations of heart muscle, edema and hemorrhage of the lung, central fatty denaturation of the liver, and congestion of many organs were histologically observed.

75-1323. Balistreri, W. F.; Partin, J. C.; Schubert, W. K. (Children's Hosp. Res. Found., Cincinnati, OH). **Hepatic necrosis following accidental chlordane exposure.** *Pediatr. Res. 7(4): 319/91; 1973.*

An infant developed vomiting and seizures, jaundice, hepatomegaly, hypoprothrombinemia, and elevated transaminases following acute, prolonged dermal exposure to liquid chlordane. Rapid clinical improvement followed exchange transfusion. Acute hepatocellular necrosis was documented by serial liver biopsy. Five days after exposure marked universal centrilobular necrosis, fatty infiltration and minimal inflammation were evident. Electron microscopy showed intact. mitochondria with large dense bodies, abundant glycogen, dilated rough endoplasmic reticulum, active Golgi apparatus, and universal fat unlike Reye's syndrome or viral hepatitis. Ten days after exposure, histology improved, triglyceride and necrosis were diminished, and mitoses were frequent. After 4 months, hepatic architecture was intact; mild triglyceride accumulation persisted; serum transaminases were normal; no clinical residua were evident. Pesticide was not present in the serum on the 5th day, nor in the adipose tissue on the 10th day. Hepatic and neurologic disease immediately following exposure suggest chlordane as the etiologic agent. The possible efficacy of exchange transfusion in suspected pesticide intoxication is emphasized.

75-1324. Doherty, R. A.; Gates, A. H. (Dep. Pediatr., Univ. Rochester Sch. Med., Rochester, NY). **Epidemic**

human methylmercury poisoning–application of a mouse model system. *Pediatr. Res. 7(4): 319/91; 1973.*

The largest known epidemic of methylmercury poisoning occurred recently in Iraq during a 2-month period in which 6,530 cases were hospitalized. There were 459 hospital deaths. The source was homemade bread prepared from seed wheat treated with a methylmercurial fungicide. Both sexes of all ages were affected. Children (1-9 yr) constituted 34% of admissions. Of 31 pregnant women who were hospitalized, 14 died. Follow-up studies of infants exposed *in utero* and by suckling are currently in progress. A mouse model has been developed for evaluating the risk to the mammalian fetus and newborn. Thus far it has been shown that: methylmercury passes freely from mother to fetus; newborn mice ingest and absorb significant amounts of mercury when suckled by mercury-dosed mothers; suckling mice excreted mercury at rates less than 1% of adult excretion rates. These observations have been useful in designing and initiating human investigations and treatment.

75-1325. Kontek, M.; Marcinkowska, B.; Pietraszek, Z. (Cardiol. Clin., Inst. Intern. Med., Acad. Med., Poznan, Poland). **Badania electroencefalograficzne u pracownikow rolnych eksponowanych na pochodne kwasow aryloalkanokarboksylowych. [Electroencephalographic studies of farm workers exposed to derivatives of arylalkane carboxylic acids.]** *Pol. Tyg. Lek. 28: 937-939; 1973.* (6 references) (Polish)

Investigations were carried out in 17 tractor drivers before and after a massive exposure to herbicides belonging to the group of derivatives of arylalkane carboxylic acid. Before the exposure, slight abnormalities were observed in the EEG tracings of 7 cases (41.3%). After 4 to 28 days of work with these agents slight disturbances of bioelectric cerebral activity were found in 11 cases (64.6%) while in 5 cases (29.5%) obvious pathological changes were observed in EEG tracings with a slowing down of the basal frequency and presence of slow (4-7/sec), high amplitude theta waves.

75-1326. Jaros, F.; Kratinova, R. (Oddelenie OUNZ, Trencin, Czechoslovakia). **Akutna otrava Birlanom 24 EC s tazkym priebehom upravena po podani TMB 4 [Acute poisoning by Birlan 24 EC with serious course cured after the application of TMB 4.]** *Prac. Lek. 25(4): 152-155; 1973.* (15 references) (Czech)

The case of an acute accidental intoxication of a 65 year old man by the organophosphate Birlane 24 EC [2-chloro-1-(2,4-dichlorophenyl)vinyl diethylphosphate, chlorfenvinphos] is reported. In addition to the symptomatologic treatment, the preparation TMB 4 was successfully used [Trimedoxime, [1,1']-trimethylene-bis] 4-[hydroxyiminomethyl]-pyridinum bromate]. The clinical picture was dominated by the nicotinic, muscarinic, and central nervous syndromes with severe breathing disorders accompanied by acidosis. Hyperglycemia passing into hypoglycemia was also described.

75-1327. Bellin, J. S.; Chow, I. (Polytechnic Inst. of New York, Brooklyn, NY). **Biochemical effects of chronic low-level exposure to pesticides.** *Res. Commun. Chem. Pathol. Pharmacol. 9(2): 325-337; 1974.* (19 references)

The biochemical effects of chronic low-level occupational exposure to dichlorvos and propham were investigated. A group of industrial workers, one third of whom were directly exposed to low levels of the pesticides, had lower values of plasma and red cell cholinesterase that did a non-industrial population. Even in the absence of clinical signs of organophosphate poisoning the cholinesterase values of the industrial workers were much lower than those of a non-worker control population. The decrease in enzyme levels was significantly correlated with subjective health complaints (sore throat and forgetfulness in particular). The symptomatic complaints of these workers who are directly exposed to the pesticide are correlated with lower values of whole blood and serum cholinesterase. The complaints were not due merely to anxiety, tension, and apprehension.

75-1328. Wakatsuki, S. (Saku Central Hospital, Usuda, Nagano, Japan). **[Mercury poisoning in Iraq due to ingestion of mercury-treated seed wheat. The author's observation and the records.]** *Rodo No Kagaku (Sci. Labor) 30(3): 48-54; 1975.* (Japanese)

Cases of poisoning by ingestion of seed grains of wheat and rice treated with alkyl-mercury fungicides were described. It was stated that there is no difference between methyl mercury and ethyl mercury from the viewpoint of toxicology. The case of poisoning by methyl mercury in Iraq was cited with the author's observation of the case after 2 years. It was pointed out that, although seed treatment with mercurial pesticides was banned in Japan in 1974, the risk due to the fate of such mercurial pesticide residues must not be neglected.

75-1329. Van Noort, H. R. (Hoofdgeneesk. Arbeidsinspect. Haren, The Netherlands). **"Population at risk" en arbeidshygienisch beleid. De expositie aan bestrijdingsmiddelen. ["Population at risk" and occupational stress. Exposure to pesticides.]** *Tijdschr. Soc. Geneesk. 51(12): 426-432; 1973.* (10 references) (Dutch)

The effects of exposure to pesticides during domestic use and during manufacture are reviewed. Levels of parathion and systox in the atmosphere and in filter masks during orchard spraying are reported. Mercury levels in urine samples taken from industrial workers in a seed disinfection plant ranged from < 10 to 170 μg/l. It is concluded that private users are exposed, but not potentially threatened, because of the low

toxicity of pesticides used in homes. However, people engaged in the manufacture of pesticides may be exposed to a potential or real threat to their health if little or inadequate attention is paid to safety and hygiene.

75-1330. Maitai, C. K.; Kamau, J. A.; Gauchi, D. M.; Njoroge, S. (Dep. Pub. Health, Pharmacol. Toxicol., Univ. Nairobi, Kenya). **An outbreak of arsenic and toxaphene poisoning in Kenyan cattle.** *Vet. Rec. 96(7): 151-152; 1975.* (5 references)

A case of poisoning was reported on May 14, 1974 in Timau, on the northern slopes of Mt. Kenya, after 260 heifers had grazed near an abandoned cattle dip last used 8 yr earlier. On the first day several animals showed restlessness, belligerence, profuse salivation, grinding of teeth, froth on the lips, and watery diarrhea. Over 50% of the animals exhibited convulsions and many became comatose just before death. A total of 70 animals showed symptoms and only 3 of these survived. The 27 which died on the first day showed gross lesions, but those that died later showed hemorrhagic gastroenteric, petechial, and ecchymotic hemorrhage on the epi- and endocardium. Lungs were emphysematous and there was plenty of froth in the trachea. The liver was slightly enlarged but gross lesions in the kidney and spleen were absent. Analysis for arsenic revealed abomasum contents, 10 mg%; liver, 50 μg%; kidney, 50 μg%; dip water, 8 mg%. Arsenic was detected in soil samples collected within 30 meters from the dip, but none was detected in soil samples collected outside this area. Concentration in soil samples ranged from 2 to 15 mg%. Toxaphene (5 to 50 mg%) was detected in soil samples within 30 meters from the dip, but none was detected in dip water. Toxaphene was also noted in gastrointestinal contents. Death was attributed to the combined effects of both arsenic and toxaphene, with toxaphene being the more important of the two poisons.

75-1331. De Sloovere, J.; Debackere, M.; Hoorens, J. (Leerst. Dierl. Pathol., NIDO, Brussels, Belgium). **Intoxicaties bij huisdieren. [Poisoning of domestic animals.]** *Vlaams Diergeneesk. Tijdschr. 40(1): 8-29; 1971.* (18 references) (Dutch)

Over the 10 year period 1960-1969, 201 cases of intoxications (1.79%) were diagnosed in 11,174 post mortems. The major one was lead intoxication (22.89%) particularly in calves. In the case of dogs the most important poison was found to be strychnine (18.9%). Chronic copper intoxication was diagnosed in cattle, pig, and sheep. Thallium particularly was found in dogs and cats. Nitrite and nitrate poisoning occurred in pigs in connection with contaminated drinking water and in cattle during the winter period due to feeding beets and grapes. Boxwood was found to be the most common poisonous plant and the most important poison in swine. Organophosphorus insecticides as well as furoxone were responsible for about 5% of the intoxication cases. Less important causes of intoxications were arsenic organochlorine pesticides, phosphorus, tylosine tartrate, dicoumarol compounds, sodium chloride, aflatoxicosis, antifreeze, cole seed, streptomycin, sodium fluoride, hexachlorophene, mercury, chlorinated naphthalenes, hydrocyanic acid, carbon monoxide, creosote, tetramisol, sulfamerazine, neomycin, heroin, chloracetamide, and badly preserved potatoes.

see also 75-1236 75-1270 75-1281 75-1300 75-1364

75-1332. Brankov, K. (Dep. Pharmacol. Toxicol., Med. Fac. Sarajevo, Sarajevo, Yugoslavia). **Uticaj antiholinesteraze i srcano-aktivnih glikozida na efekte stimulacije spoljnih nerava in vitro. [The influence of anticholinesterase and cardiac active glycosides upon the effect of extrinsic nerve stimulation in vitro.]** *Acta Med. Iugoslav. 26: 415-420; 1972.* (12 references) (Serbo-Croatian)

Since organophosphorus compounds (OPhC) act upon smooth and striated muscles, the influence of cardiotonics before and after poisoning with Neguvon (trichlorfon) in vitro was investigated. The cardiac-active glycosides used were 0.02% K-Strophantin-K, 0.02% Lanatoside C, 0.05% Digitonin and 0.01% Acylanid. The rat isolated innervated urinary bladder was used as a model system for the smooth muscle while the rat isolated innervated esophagus was used as a model system for the striated muscle. Cardiac-active glycosides in a concentration of $5 \times 10^{-3} M$, in a great number of cases, did not have any effect on the intensity of micturition contraction and the tonus of the detrusor. Similarly, cardiotonics did not reduce the contractions and did not change the tonus of the esophagus. Neguvon in a concentration of 10^{-3} intensified the effect of the pelvic nerve stimulation accompanying the increased tonus of the detrusor. The same concentration of Neguvon reduced the effect of the cholinergic nerve stimulation and the contractions of the esophagus. When cardiac-active glycosides were added to the isolated organ bath after Neguvon, they acted just like Neguvon itself. The cardiac-active glycosides Strophanthin-K, Lanatoside C, Acylanid, and digitoxin intensified the effect of nerve stimulation of the isolated urinary bladder and esophagus in the presence of Neguvon.

75-1333. Rump, S.; Rabsztyn, T.; Kopec, J. (Dep. Toxicol., Military Inst. Hyg. Epidemiol., Warsaw, Poland). **Effects of cholinesterase inhibition on the visual evoked potentials in the rabbit and their modification with various drugs.** *Activ. Nerv. Super. 16(3): 224-225; 1974.*

The effects of drugs on visual evoked potentials (VEP) when organophosphates were given were studied in regard to the central effect of anticholinesterases and their antagonists. Twenty-five rabbits in which skull electrodes had been implanted over the visual cortex were watched after inhibition of cholinesterase with fluostigmine, atropine sulfate, ethylbenztropine bromide, diethazine hydrochloride, and diazepam hydrochloride. ChE activity in the brain after fluostigmine administration (0.3 mg/kg) was 55% that of controls. The VEP registered flat with every component decreased. Subsequent administration of the test drugs changed this picture. Atropine and ethylbenztropine had the most remarkable effects on the most sensitive part of the VEP. Diethazine had an action most nearly approaching normalization; however, this was a transient effect which disappeared within 10 min. Diazepam, though it increased component V to the highest value and component VIII to a normal level, caused the fall of the amplitude of component XI. These results indicate that the decrease of amplitude of VEP is observed earlier than the desynchronization of cortical activity. The effect of cholinolytics on the abnormalities of VEP induced by ChE inhibition are clearest, as is understandable, but it is surprising that diethazine has an effect which looks like near complete normalization of VEP, though it possesses no action on cortical activity.

75-1334. Shoemaker, J. P.; Bradley, R. L.; Hoffman, R. V., Jr. (Res. Serv., VA Hosp., Huntington, WV 25701). **Direct injection of experimental mammary adenocarcinoma with picloram.** *Am. J. Med. Sci. 268(6): 333-335; 1974.* (2 references)

The effect of a subcutaneous injection of 0.25 ml picloram into mammary adenocarcinoma in 10 adult C3H mice was investigated. The picloram was injected daily directly into the tumors as soon as they became palpable (± 10 days). The tumors of the control group were injected directly each day with 0.25 ml saline. The mean tumor size in cm^3 was determined on alternate days. The experimental mice tumors were statistically smaller than the control mice tumors on seven of eight check days ($p < 0.05$). The difference between the mean survival time of the experimental mice (34.9) days and the control mice (26.7 days) was statistically significant ($p < 0.025$).

75-1335. Skromne-Kadlubik, G.; Alvarez-Cervera, J.; Cortes-Marmolejo, F. (Serv. Med. Nuclear, Cent. Hosp. "20 de Noviembre", Mexico City, Mexico). **Gamagrafia suprarrenal en seres humanos utilizando ^{131}I-dicloro difenil-dicloroetano. [Human suprarenal gammagrams using DDD labeled with I-131.]** *Arch. Inst. Cardiol. Mex. 43: 245-248; 1973.* (10 references) (Spanish)

Suprarenal (adrenal) gammagrams were taken on a group of 13 normal human volunteers, using DDD (TDE) labeled with I-131 according to a method previously described in order to obtain information on the normal image which should be seen in this type of study. The results indicated that there are variations in the image in different individuals and also in the same individual at different hours of the day (circadian rhythm). The same study was also performed on five patients with a clinical diagnosis of suprarenal tumors (two cortical adenomas and three pheochromocytomas) made by indirect methods (urography, tomography, etc.). It was possible to demonstrate the size and exact location of the tumor by direct visualization of the suprarenal gland, demonstrating that the method is very useful for the diagnosis of tumoral diseases of the suprarenal cortex or medulla that affect its size or position. No toxicity nor radiotoxicity was found at the doses used in our studies. The method is practical, simple, without risks, and can be used for diagnostic purposes in the medical clinic.

75-1336. Rodionov, G. A. (Lab. Pathol. Morphol. Histochem., All-Union Sci. Res. Inst. Hyg. Toxicol. Pesticides, Polymers, Plastic Materials, Kiev, USSR). **K probleme izucheniya patogennogo vliyaniya khimicheskikh veshchestv vneshney sredy na organizm. [On the problem of investigating the pathogenic effect of environmental chemical substances on the organism.]** *Arkh. Patol. 36(10): 80-89; 1974.* (83 references) (Russian)

Studies on the pathological effects of environmental chemicals, including pesticides, on the human and animal organism are reviewed. Apart from the general toxicity, the allergenic, blastomogenic, embryotoxic, teratogenic, and immunosuppressive effects of pesticides should also be studied. Zineb and maneb were found to cause adenoma of the lungs in mice, while dithiocarbamates, especially urea derivatives, were determined to be thyrotropic in their action. Organophosphorus compounds and DDT are hepatotropic substances par excellence. Inhibition of the cholinesterase activity, hemodynamic disorders, and destructive, necrotic, and even reactive processes were observed in animals poisoned with organophosphorus pesticides. Carbaryl and DDT aggravated the course of cholesterol-induced atherosclerosis in rabbits, and trichlorfon intensified and accelerated the pathological changes induced by carbon tetrachloride in rats.

75-1337. Lake, B. G.; Longland, R. C.; Hodgson, R. A.; Severn, B. J.; Gangolli, S. D.; Lloyd, A. G. (British Ind. Biol. Res. Assoc., Carshalton, England). **The effect of some inducers of hepatic xenobiotic-metabolizing enzymes on the urinary excretion of D-glucuronic acid metabolites in the rat.** *Biochem. Soc. Trans. 3(1): 188-191; 1975.* (13 references)

A sensitive GLC procedure was developed for the determination of D-glucaric acid and a number of intermediates of the D-glucuronic acid pathway in human and animal urine. The effect of treatment with some model compounds which induce microsomal enzymes in white male Wistar rats on the urinary excretion of components of the D-glucuronic acid pathway is investigated and the results are presented. Dichlorodiphenyltrichloroethane, a chlorinated hydrocarbon pesticide, was administered i.p. at 100 mg/kg/day for 7 days. The pesticide significantly increased the relative liver wieght, cytochrome P-450 content, and the activities of aniline 4-hydroxylase and 4-methylumbelliferyl glucuronyltransferase. This pesticide did not significantly enhance the excretion of D-glucaric acid, but did enhance the excretion of both L-gulonic acid and xylitol. This study indicates that the measurement of a spectrum of metabolites of the D-glucuronic acid pathway provides a more reliable index of the induction of hepatic microsomal xenobiotic-metabolizing enzymes than the determination of a single urinary component such as D-glucaric acid.

75-1338. Suzuki, H.; Ishikawa, S. (Dept. Ophthalmol., Kitasato Univ. Sch. Med., Sagamihara City, Kanegawa, Japan). **Ultrastructure of the ciliary muscle treated by organophosphate pesticide in beagle dogs.** *Br. J. Ophthalmol. 58(11): 931-940; 1974.* (37 references)

Ultrastructural changes in the ciliary muscle in 5 beagle dogs treated by the organophosphate compound, ethylthiometon (5-15 mg/dog/day, 5 days/week for 2 yr, p.o.) have been investigated. All dogs receiving ethylthiometon showed myopia 12 months after administration which progressed until cessation of the drug. In general, higher doses produced more severe myopia. Marked ultrastructural changes were noted in the ciliary muscle cells of a treated dog with about 1 D myopia. The cytoplasm in some muscle cells was occupied exclusively with unique membranous structures which were composed of numerous paired membranous structures. The outer spaces of each pair were filled with an amorphous mass, highly distended, and occasionally cystic. The interspaces were closely apposed and contained a small amount of cytoplasm which was continuous with the peripheral cytoplasm. Further destructive changes were noted in the ciliary muscle of the high dosage group with maximum myopia. The cytoplasm in about half the cells was occupied with a large amount of amorphous mass and had scarcely any definite organelles. Only a few vacuolar systems, mitochondria, and unique membranous structures were preserved. In spite of the severe destruction within the muscle cells, the cell membrane of the muscle cell and the axon and end-plate of the nervous system showed no remarkable change. It is noted that normal cells were observed among affected cells and they were intermingled. This would indicate that there may be two kinds of cells in the ciliary muscle, which differ substantially in their sensitivity to cholinesterase inhibitors.

75-1339. Chow, A. Y. K.; Murphy, S. D. (Dep. Physiol., Kresge Cent. Environ. Health, Harvard Univ., Sch. Pub. Health, Boston, MA 02115). **Production of a methemoglobin-forming metabolite of 3,4-dichloroaniline by liver in vitro.** *Bull. Environ. Contam. Toxicol. 13(1): 9-13; 1975.* (11 references)

A convenient assay method for determining the capacity of liver homogenates to metabolize 3,4-dichloroaniline (DCA) to an active methemoglobin-forming metabolite has been developed. Whole liver homogenates were incubated with DCA, a NADPH-generating system, and heparinized rat blood, which served as the source of hemoglobin, the reactant with the active metabolite of DCA. The activity was measured in terms of the quantity of methemoglobin formed. Using this method, the relative activities of livers from male and female mice, rats, and guinea pigs were compared. Mouse and guinea pig livers were about equally active in producing methemoglobin-forming metabolite from DCA, while rat liver was less effective. Mouse and guinea pig livers showed no sex differences in this respect, but male rat livers were significantly more active than female rat livers. When heparinized male rat, mouse, and guinea pig

blood were used in the incubation mixture, the percent methemoglobin obtained was highest with the rat blood followed by the guinea pig and mouse blood; these differences appeared to be due to different rates of methemoglobin reduction. Thus, heparinized rat blood is preferred in the assay method.

75-1340. Reddy, G.; Khan, M. A. Q. (Dep. Biol. Sci., Univ. Illinois, Chicago, IL 60680). **Fate of photodieldrin under various environmental conditions.** *Bull. Environ. Contam. Toxicol. 13(1): 64-72; 1975.* (16 references)

Primary leaves of bush red kidney beans were treated with 2 g of ^{14}C-photodieldrin (PD, the metabolic product of the photo-metabolites of aldrin and dieldrin) and were kept varying lengths of time under artificial light and sunlight. After different time intervals, the chlorinated hydrocarbon residues on the leaves were extracted and analyzed by liquid scintillation and TLC autoradiography. With increasing time, the ^{14}C-PD slowly but steadily dissipated on the leaf surface under both lighting conditions; the loss of PD from the leaf surface was 50% after 1 day and 85-90% after 8 days. PD appears to be either metabolized by the plant after being absorbed into the leaf, or volatilized from the surface. One metabolic product was found in leaves exposed to sunlight, but not artificial light, for 2-4 days. ^{14}C-PD was considerably stable on bean leaves stored at -20°C; small losses due to volatilization occurred and there were no metabolites formed. In a second experiment, ^{14}C-PD was added to growing cultures of freshwater algae. Under laboratory conditions, ^{14}C-PD was persistent in the water with and without algae. About 40% of the added PD was adsorbed or absorbed by the algae during this period. There were no detectable metabolites in either water or organisms. In a third experiment, ^{14}C-PD was spotted on silica gel plates which were kept under UV or room light, in the dark, or in a refrigerator. About 90-95% of the applied PD was recovered from plates stored for 1 month in the refrigerator or under laboratory light; no metabolic products were detected. After 12 hours under UV light, PD formed two metabolic products; the concentrations of PD and metabolites decreased with time up to 30 days.

75-1341. Butler, G. L.; Deason, T. R.; O'Kelley, J. C. (Life Sci. Div., Syracuse Univ. Res. Corp., Syracuse, NY 13210). **Loss of five pesticides from cultures of twenty-one planktonic algae.** *Bull. Environ. Contam. Toxicol. 13(2): 149-152; 1975.* (4 references)

The uptake and metabolism of five pesticides by actively growing cultures of planktonic algae were studied. Atrazine, carbaryl, and diazinon were added to the cultures at a rate of 1 ppm, and methoxychlor and 2,4-D were added at 0.01 ppm. After 2 weeks, the cultures were extracted and the extracts analyzed by gas-liquid chromatography. Thirty-six planktonic isolates were studied, of which 21 appeared to represent separate taxonomic or physiological entities. About 80% of the carbaryl added was broken down in a nonbiological fashion within the first week. Essentially all of the atrazine was recovered from the 12 isolates capable of limited growth at 1 ppm. Only 62% of the diazinon was recovered from one isolate, 75-96% being recovered from the other 20. Substantially less methoxychlor was recovered (20-79%), and less than 20% of the total 2,4-D added was recovered from seven isolates. Thus, although algae do not appear capable of removing atrazine from the medium, several of the isolates were able to remove diazinon, 2,4-D, and/or methoxychlor.

75-1342. Desaiah, D.; Koch, R. B. (Dep. Biochem., Mississippi State Univ., Mississippi State, MS 39762). **Inhibition of ATPases activity in channel catfish brain by Kepone and its reduction product.** *Bull. Environ. Contam. Toxicol. 13(2): 153-158; 1975.* (19 references)

The effects of chlordecone (Kepone) and its reduction product, decachloropentacylo-decan-5-ol (DCPD), on channel catfish brain ATPases were studied in vitro. Of the brain ATPases, oligomycin-sensitive Mg^{2+} ATPase (mitochondrial) showed the greatest sensitivity to Kepone and DCPD, the inhibition by both compounds being similar. The inhibition of Na^{+}-K^{+} ATPase by Kepone and DCPD was also quite similar and the degree of inhibition increased with increasing pesticide concentration. Oligomycin-insensitive Mg^{2+} ATPase activity showed a similar sensitivity to Kepone and DCPD, although the inhibition was less than that observed with mitochondrial Mg^{2+} ATPase. In all cases, the inhibition was highly significant. The data suggest that the initial toxic action of Kepone and DCPD may be a reduction of ATP synthesis via inhibition of oxidative phosphorylation in the mitochondria.

75-1343. Borthwick, P. W.; Tagatz, M. E.; Forester, J. (U.S. Environ. Protect. Agency, Gulf Breeze Environ. Res. Lab., Sabine Island, Gulf Breeze, FL 32561). **A gravity-flow column to provide pesticide-laden water for aquatic bioassays.** *Bull. Environ. Contam. Toxicol. 13(2): 183-187; 1975.* (5 references)

A column containing granular pesticide, bait, or inert material coated with pesticide may be utilized to achieve realistic concentrations of pesticides in assay water without using a solvent. Mirex was introduced into flow-through aquatic bioassay systems without a solvent by means of a gravity-flow column containing mirex bait. The bioassay systems consisted of three tanks, each receiving filtered tap water which had passed through 150 grams of mirex bait; three additional tanks received effluent from columns containing 150 grams of control bait. Biweekly water samples from the tanks were analyzed by electron-capture gas chromatography. The concentrations of mirex among the individual tanks in each test were not statistically significant, whereas the concentrations did differ significantly between different

tests. Fluctuations in the mirex concentrations within individual tanks were not significant. The gravity-flow column is being utilized in seasonal tests to deliver mirex-laden water to determine the toxicity and uptake of mirex by several animal species in an artificial estuarine ecosystem.

75-1344. Truhaut, R.; Pham-Huu-Chanh; Van Haverbeck, G.; Azum-Gelade, M. C.; Saint Ruf, G.; Lareng, L. (Lab. Toxicol. UER, Univ. Rene Descartes, Paris, France). **Toxicite a court terme de la tetrachloro 2,3,7,8 dibenzo *p*-dioxine chez le rat: etude structurale, ultrastructurale et enzymologique du foie. [Acute toxicity of tetrachlorodibenzo-*p*-dioxin in rats: structural, ultrastructural, and enzymological study of the liver.]** *C. R. Hebd. Acad. Sci. Ser. D 279: 1565-1569; 1974.* (8 references) (French)

Structural, ultrastructural, and enzymatic studies of the liver following short-term poisoning of male and female Wistar rats with 2,3,7,8-tetrachlorodibenzo-*p*-dioxin are described. The animals were administered 50 μg/kg doses p.o. or 100 μg/kg doses i.p. daily, five days a week, for 24 days. After the first death occurred on the 8th day of the experiment, the mortality increased to 80% by the 22nd day. The liver, showing normal macroscopic appearance, exhibited the following microscopic changes: disappearance of the normal trabecular structure of the liver parenchyma, appearance of pseudoacinous structures, and aggregation of hepatocytes around canaliculi or lacunae. Biochemical investigations revealed nearly total absence of adenosine triphosphatase in the pericanalicular region. The appearance of pseudoacinous structures, and the disappearance of the pericanalicular adenosine triphosphatase suggest the potential carcinogenicity of dioxin. The hepatocyte mitochondria maintained their functional and morphological integrity.

75-1345. Korn, S.; Earnest, R. (Natl. Marine Fish. Serv., Tiburon Fish. Lab., Tiburon, CA 94920). **Acute toxicity of twenty insecticides to striped bass, *Morone saxatilis*.** *Calif. Fish Game 60(3): 128-131; 1974.* (8 references)

The acute toxicity of twenty insecticides to juvenile striped bass was determined in saline water. All bioassays were 4-day tests, using proportional diluters as described earlier. Endrin, endosulfan, DDT, and dursban were the most toxic with median lethal concentration values of less than 1 μg/l. Carbaryl and abate (temophos) were the least toxic with median lethal concentration values over 1,000 μ/l. TDE, heptachlor, methoxychlor, toxaphene, aldrin, lindane, chlordane, malathion, parathion, dieldrin, EPN, fenthion, dibrom, and methyl parathion were also tested; these are arranged in order of decreasing toxicity. The intermittent flow bioassay data supplied by this study is useful in screening out the more toxic compounds from those in use or being considered for application. The determination of acute toxicity is the first step in evaluating chronic sub-lethal effects of compounds potentially harmful to aquatic organisms.

75-1346. Vargova, M.; Kovalcik, V. (Vyskumny Ustav Hyg., Bratislava, Czechoslovakia). **K otazke hygienicko-toxikologickeho hodnotenia pesticidnych latok. [Hygienic-toxicological evaluation of pesticides.]** *Cesk. Hyg. 18(3): 110-113; 1973.* (4 references) (Czech)

The s.c. median lethal time (LT50) (defined as the time within which 50% of the animals die) may serve as an indicator of toxicity of a dose of repeatedly administered heptachlor. Logarithm of the median lethal time proved to be linearly dependent on the dose according to the relation: $\log LT50 = \log C - n \cdot \log D$. The value of the constant "n" appears to be particularly important for an evaluation of safety as it is dependent not only on the character of the compound but also on the frequency of administration.

75-1347. Yarbrough, J. D.; Coons, L. B. (Dep. Zool., Mississippi State Univ., Mississippi State, MS 39762). **A comparative cytological study between hepatocytes of insecticide-resistant and susceptible mosquitofish (*Gambusia affinis*).** *Chem.-Biol. Interactions 10(4): 247-254; 1975.* (14 references)

The histology and ultrastructure of hepatocytes from insecticide-resistant mosquitofish (*Gambusia affinis*) were investigated and compared to susceptible mosquitofish. In the hematoxylin-eosin prepared liver material from insecticide-resistant mosquitofish the cells were greatly enlarged in comparison to cells from susceptible mosquitofish. The insecticide-resistant hepatocyte profile is 38.7 ± 0.40 micron times 28.9 ± 0.37 micron as compared to 22.3 ± 0.30 micron times 15.8 ± 0.26 micron for hepatocytes from susceptible mosquitofish. Measurements were made with a scanning electron microscope. When hepatocytes from the two fish preparations are compared by a t-test, there is a highly significant difference at the $P < 0.001$ level. Hepatocyte nuclei from resistant and susceptible mosquitofish preparations appear normal at both the light and electron microscopic level. The most obvious subcellular difference is a heavy lipid inclusion in hepatocytes from resistant fish which remained essentially unchanged in mosquitofish starved for up to 2 wk. No observable differences were noted in any cellular organelles.

75-1348. Makhija, S. J.; Pawar, S. S. (Div. Biochem., Dep. Chem., Marathwada Univ., Aurangebad, Maharashtra, India). **Alterations in hepatic drug metabolism and lipid peroxidation during administration of Baygon, a pesticide.** *Chem.-Biol. Interactions 10(4): 295-299; 1975.* (20 references)

The effect of Baygon (propoxur) on the liver microsomal drug-metabolizing enzyme system and lipid peroxidation of young male albino CF rats was investi-

gated. Injections were made for 2 days at a dose level of 5 mg/kg (low) and 25 mg/kg (high), while control animals received a placebo. There was a slight, but significant increase in liver weights after acute treatment, while chronic treatment decreased the liver weights slightly. The relative liver weights slightly increased with low dose and significantly increased with high dose. The decreases in aminopyrine *N*-demethylase activities were 9.7 and 19.7% with low and high dose respectively. The decrease in acetanilide hydroxylase activity was only 1.6% with low dose, but was 22.3% with high dose. Low doses significantly increased enzymatic lipid peroxidation, but slightly increased non-enzymatic lipid peroxidation. The increase in lipid peroxidation was more significant with high dose. The animals showed physical changes such as salivation and fasciculations. Toxic effects leading to death were noted when the animals were injected with a dose of propoxur above 25 mg/kg.

75-1349. Parlar, H.; Gaeb, S.; Lahaniatis, E. S.; Korte, F. (Inst. Oekol. Chem., Univ. Munich, Freising-Weihenstephan, Germany). **Beitraege zur oekologischen Chemie XCIII. Synthese und analytisches Verhalten von 1-endo-Hydroxychlorden und 1-exo-Hydroxychlorden. [Contribution to ecological chemistry. Part 93. Synthesis and analytical behavior of 1-endo-hydroxychlordene and 1-exo-hydroxychlordene.]** *Chemosphere 1: 15-20; 1975.* (10 references) (German)

1-Hydroxychlordene is known as a transformation product of heptachlor in plants and soil. Formation of 1-hydroxy-2,3-epoxychlordene, the terminal metabolite of heptachlor in rats, may take place via 1-hydroxychlordene. Chemical formation of 1-hydroxychlordene has been proposed in addition to the enzymatic means of formation. The synthesis and analytical behavior of 1-endo-hydroxychlordene and 1-exo-hydroxychlordene were studied. Studies on the synthesis with lithium-aluminum tetradeuteride revealed a reaction mechanism which begins with a non-stereospecific nucleophilic attack of deuterium on the β-carbon atom of the unsaturated ketone obtained by the oxidation of 1-exo-hydroxychlordene with *N*-bromosuccinimide according to Barakat's method. The reaction of the unsaturated ketone with lithium-aluminum hydride in ether results in 1-endo-hydroxychlordene following hydrolysis.

75-1350. Dvorchik, B. H.; Maren, T. H. (Dep. Obstet. Gynecol., M. S. Hershey Med. Cent., Hershey, PA 17033). **Distribution, metabolism, elimination and toxicity of ^{14}C-DDT in the dogfish, *Squalus acanthias*.** *Comp. Gen. Pharmacol. 5(1): 37-49; 1974.* (41 references)

The dynamic movement and toxicity of DDT in a representative elasmobranch, the dogfish *Squalus acanthias*, following i.v. dosing of DDT was studied. The drug was rapidly distributed to all organs, with a final redistribution to liver and muscle. DDT was associated with plasma lipoproteins. This association, plus its low water solubility, may explain the lack of any binding to erythrocytes. Liver and muscle are the principal storage sites for lipid in elasmobranches. While the total amount of lipid in these organs was about the same, the concentration of lipid in the liver was about 5-fold that in muscle. The ratio of DDT concentration in liver lipid to that in muscle lipid was about 2:1 throughout the study. Due to the low water solubility of DDT and its affinity for plasma proteins, diffusion across the gills did not occur. Excretion of DDT by the gills may have occurred too slowly to be measured. Excretion by the bile and urine was also negligible. Analysis of the radioactive species present in the liver during a 2-week period following administration of ^{14}C-DDT indicated that at least 95% of the drug present in the liver was the parent compound. The i.v. dose which produced about 50% mortality within 24 hr was 10 mg/kg. Studies on the concentration of DDT in the brains of dogfish that died following intravascular administration of 10 mg/kg DDT suggested that the brain of elasmobranches may be more sensitive to DDT than that of mammals.

75-1351. Kawatski, J. A.; McDonald, M. J. (Dep. Biol., Viterbo Coll., La Crosse, WI 54601). **Effect of 3-trifluoromethyl-4-nitrophenol on in vitro tissue respiration of four species of fish with preliminary notes on its in vitro biotransformation.** *Comp. Gen. Pharmacol. 5(1): 67-76; 1974.* (17 references)

The effect of TFM on fish tissue in vitro respiration, as measured by oxygen consumption, was studied. The fish species and tissues examined were: liver of white sucker (*Catostomus commersoni*), rainbow trout (*Salmo gairdneri*), largemouth bass (*Micropterus salmoides*), bluegill sunfish (*Lepomis macrochirus*) and brain of rainbow trout. TFM (10.0 ml/l) caused a significant reduction in oxygen consumption by homogenates of the tissues examined. Largemouth bass and white sucker livers were most sensitive, showing significant reduction in oxygen utilization also at 1.0 mg/l TFM. In the presence of 1.0 mg/l TFM, the rate of respiration by trout brain increased significantly, but at 0.5 mg/l there was no significant change in respiration rate. No significant biotransformation of TFM was noted in trout brain homogenates, but all other fish tissues produced at least 2 TFM metabolites when exposed to 1.0 or 10.0 mg/l (^{14}C)TFM.

75-1352. Willis, D. E.; Addison, R. F. (Bedford Inst. Oceanogr., Dartmouth, Nova Scotia, Canada). **Hydroxylation of biphenyl in vitro by tissue preparations of some marine organisms.** *Comp. Gen. Pharmacol. 5(1): 77-81; 1974.* (10 references)

Biphenyl was metabolized in vitro to 4-hydroxybiphenyl and, to a lesser extent, to 2-hydroxybiphenyl by tissues of *Raja ocellata, Salvelinus fontinalis, Homarus americanus, Gerydon quinquidens, Cancer irroratus,*

Mytilus edulis, *Asterias vulgaris*, and by zooplankton. Rates of 4-hydroxybiphenyl production ranged from 400 nmoles/g tissue/hr in *Raja ocellata* to about 2 nmoles/g tissue/hr in *Asterias vulgaris*. This data indicates that the capacity to metabolize biphenyl mainly to the 4-hydroxy and also to the 2-hydroxy derivatives existed in all tissue samples studied. The wide range of 4-hydroxylation activity observed in various studies of various species suggests some real differences among those species, and further suggests that in general the higher rates are associated with more complex organisms. Hydroxylation in the 2-position was, in general, slower than in the 4-position. The 4-hydroxylation rates noted in this study were well below those observed in terrestrial species such as the rabbit, rat, mouse, hamster, and guinea pig. It is emphasized that the data presented describes the metabolism of biphenyl in an artificial system and does not necessarily indicate the hydroxylation rates which will be found in vivo.

75-1353. Johannsen, F. R.; Knowles, C. O. (Dep. Entomol., Univ. Missouri, Columbia, MO 65201). **Toxicity and action of fluenethyl acaricide and related compounds in the mouse, housefly and twospotted spider mite.** *Comp. Gen. Pharmacol. 5(1): 101-110; 1974.* (29 references)

The toxicity and action of fluenethyl and related compounds in mice, houseflies, and twospotted spider mites were investigated. Toxicity varied markedly with the test compound, method of application, and biological species. Monofluoroacetic acid, monofluoroethanol, and fluenethyl were the most active materials examined. Intoxicated mice and houseflies exhibited classic symptoms of organofluorine poisoning. Spider mites exhibited a brief period of hyperactivity. Ethanol, isoniazid, triazole, acetamide, sodium acetate, and monoacetin inhibited the toxicity of fluenethyl and monofluorethanol to mice. The metabolic transformation of these compounds may be essential for toxicity. Mice, houseflies, and spider mites poisoned with fluenethyl and other fluorine-containing compounds accumulated high levels of citrate as compared to controls, indicating that aconitase or citrate (isocitrate) hydrolyase was attacked. The monofluoroethanol released upon esteratic cleavage of fluenethyl was oxidized to monofluoroacetic acid, which inhibited aconitase.

75-1354. Singh, G.; Pant, N. C. (Indian Agric. Res. Inst., New Delhi, India). **Degradation of insecticides by the cultured symbiotes of *Cletus signatus* Walker (Coreidae: Heteroptera).** *Curr. Sci. 43(19): 624-625; 1974.* (4 references)

Studies were conducted to establish the role of symbiotic microorganisms in the degradation of DDT, parathion, and carbaryl. Symbiotes of *C. signatus* rapidly degraded each pesticide when these were incubated with culture. Degradation was more pronounced at 6 and 24 hr after incubation started; at these times 820 and 850 μg DDT, 20 and 60 μg parathion, and 18 and 22 μg of carbaryl were degraded respectively. Utilization of the pesticide after 24 hr was negligible. *B. cereus* generally starts sporulating by 24 hr and this may be the cause of the near stoppage of the metabolism of the pesticides. The value of this characteristic of symbiotes to the host insect can be determined by the in vivo studies on the comparative susceptibility of the aposymbiotic and normal bugs to the insecticides.

75-1355. Lee, K. C.; Rao, G. M.; Barnett, F. L; Liang, G. H. (Dep. Agron., Kansas State Univ., Manhattan, KS 66506). **Further evidence of meiotic instability induced by atrazine in grain sorghum.** *Cytologia 39(4): 697-702; 1974.* (15 references)

Evidence of meiotic disturbance in grain sorghum following application of atrazine as a preemergence herbicide is reported. Two pure lines of sorghum were planted. Atrazine was applied in aqueous solution at 20 gal/A (1871/ha). The concentration of the solution was adjusted to give these rates of active ingredients: O lb/A (control), 3 lb/A (3.3 kg/ha), 6 lb/A (6.6 kg/ha), and 9 lb/A (10 kg/ha). Cytological observations indicated that low frequencies of meiotic aberrations occurred in all populations treated with atrazine while no similar findings occurred in controls. There appeared to be no clear relation between rate of application and frequency of meiotic irregularity. Frequency of total aberrations seemed higher in the F_1 than in the F_2 or parental lines; irregularities seemed more frequent in Combine 7078 than in Combine Kafir 60. More common aberrations were aneuploidy, polyploidy, metaphase I univalents, and multinucleate cells. Anaphase I bridges and telophase I laggards were less frequent. A primary cause for concern growing out of these findings is that irregularities of the kind observed may enhance development of off types and thereby create problems of genetic instability in lines or cultivars. The possibility that atrazine has similar effects on weeds is also worrisome. It is stressed that these abnormalities were found only rarely and that further research is needed.

75-1356. Fabacher, D. L.; Chambers, H. (Entomol. Dep., Mississippi State Univ., Mississippi State, MS 39762). **Resistance to herbicides in insecticide resistant mosquitofish.** *Environ. Lett. 7(1): 15-20; 1974.* (5 references)

A study was conducted to determine the relative toxicity of herbicides to insecticide-susceptible mosquitofish and to determine any differences in susceptiblity of insecticide-susceptible and resistant mosquitofish to the more toxic herbicides. The percent mortality of insecticide-susceptible mosquitofish treated with 10 ppm of selected herbicides was 0-10% for diuron, fluometuron, MSMA, DSMA, chloramben, naptalam, Ordram (molinate), Herban (norea), 2-4-D acid, 2,4-D

dimethylamine salt, and 2-4-DB dimethylamine salt; 10-50% for monuron, nitralin, dalapon, prometryne, Vernam, and linuron; and 50-100% for propanil, trifluralin, DNBP, 2,4-D isopropyl ester, 2-4-D *N*-butyl ester, 2,4,5-T acid, and 2,4,5-T butoxyethanol ester. Insecticide-resistant fish showed a 1.87 X resistance to 2,4-D butyl ester and a 2.05 X resistance to trifluralin. There was essentially no difference in the toxicity of DNBP to the two populations of fish. The LC50 values for susceptible and resistant fish treated with DNBP were 0.87 and 0.96; with trifluralin, 2.00 and 4.10; and with 2,4-D butyl ester, 0.91 and 1.70 respectively. It is not likely that the noted resistance to 2,4-D ester or trifluralin resulted from direct exposure to these materials, but rather represents an indirect effect of resistance to insecticides. This study indicates that environmental toxicants can alter the basic physiology of an animal species to change its toxic response to other pollutants such as herbicides.

75-1357. Nicholson, S. A.; Clerman, R. J. (Dep. Biol., State Univ. College, Fredonia, NY 14063). **Toxicity of diquat to the crustacean amphipod *Hyalella* from Chautauqua Lake.** *Environ. Lett. 7(1): 215-227; 1974.* (13 references)

The sensitivity of *Hyalella* from Chautauqua Lake to diquat was determined under standard laboratory conditions. The crustaceans were very sensitive to diquat concentrations commonly used for weed control. The median 24 hr tolerance limit of *Hyalella* sp. to diquat in lakewater was 1.26 ppm. The marked decline in numbers of *Hyalella* sp. in Chautauqua Lake over the past few decades could be related to its extreme sensitivity to herbicides such as diquat, not only via direct toxicity but also through indirect effects such as habitat destruction and food chain contamination.

75-1358. Neville, G. A.; Berlin, M. (Health Prot. Branch, Health Welfare Canada, Tunney's Pasture, Ottawa, Ontario, Canada). **Identification and biotransformation of organomercurial compounds in living systems: a review of current understanding.** *Environ. Res. 7: 75-82; 1974.* (46 references)

The chemical basis for mercurial toxicity in various living systems is the overall concern of this review. Biotransformation of organomercurial forms of special concern to environmentalists and structural evidence for derived mercurial moieties are discussed in the perspective of evolving understanding of biotransformation and methodological development. Identification of the mercurial forms in tissues, body fluids, and cellular bodies is a necessary, but challenging, key to the understanding of mercurial toxicology. (Author abstract by permission of Academic Press)

75-1359. Bachofer, R.; Lingens, F.; Schafer, W. (Inst. Microbiol. Molekularbiol., Univ. Hohenheim, Stuttgart, Germany). **Conversion of aniline into pyrocatechol by a *Nocardia* sp.; incorporation of oxygen-18.** *FEBS (Fed. Eur. Biochem. Soc.) Lett. 50(2): 288-290; 1975.* (16 references)

In a study aimed at further breaking down pesticide degradation products, the mutant AM 144, isolated after treatment of the *Nocardia* wildtype strain A with *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine, was grown in glucose minimal medium supplemented with aniline. The cultures were incubated in a nitrogen atmosphere or in flasks to which 100 ml of oxygen-18 gas had been added. Detectable amounts of pyrocatechol were not produced after incubation in nitrogen. However, in the $^{18}O_2$-labeling experiment, the mass spectrum of the isolated accumulate showed a parent peak at m/e 114 which corresponds to double-labeled pyrocatechol. A small peak at m/e 110 was in accord with the formation of pyrocatechol from the low percentage of $^{16}O_2$ in the medium. Only a negligible amount of part-labeled pyrocatechol was formed, indicating that two atoms of oxygen were incorporated at the same time into the aromatic nucleus.

75-1360. Lui, H.; Sweeney, G. D. (Pharmacol. Group, McMaster Univ., Hamilton, Ontario, Canada). **Hepatic metabolism of hexachlorobenzene in rats.** *FEBS (Fed. Eur. Biochem. Soc.) Lett. 51(1): 225-226; 1975.* (5 references)

Hexachlorobenzene (HCB) was mixed with powdered rat chow to a concentration of 0.25% in the diet of male Long Evans rats. Thin-layer chromatography identified a single significant spot in the urine of HCB treated rats which was absent from control animals, and had a refractive index matching that of pentachlorophenol. Gas chromatography similarly yielded a peak with retention time matching that of pentachlorophenol. Combined gas chromatography-mass spectrometry confirmed the findings. In addition to pentachlorophenol, more polar compounds presumed to be derivatives of HCB were also detected. This finding probably demonstrates an early stage in the HCB metabolism, i.e., the introduction of a single OH group. While HCB has low acute toxicity, pentachlorobenzene has an oral LD50 in rats of 180 mg/kg. Formation of pentachlorophenol from HCB may proceed either through a free-radical mechanism or by initial formation of an arene oxide. In either event, reactive intermediates may form covalent bonds with cellular constituents leading to irreversible cell damage.

75-1361. Hook, J. B.; Bailie, M. D.; Johnson, J. T.; Gehring, P. J. (Dep. Pharmacol., Michigan State Univ., East Lansing, MI 48823). **In vitro analysis of transport of 2,4,5-trichlorophenoxyacetic acid by rat and dog kidney.** *Food Cosmet. Toxicol. 12(2): 209-218; 1974.* (14 references)

Renal transport of 2,4-5-trichlorophenoxyacetic

acid (2,4,5-T) was studied quantitatively in vitro using renal cortical slices from dogs and rats, in an attempt to explain species differences in the biological half-life of the compound. Addition of 2,4,5-T to slices of rat renal cortex competitively inhibited active transport of *p*-aminohippuric acid without altering transport of the organic cation, *N*-methylnicotinamide. Renal cortical slices from rats and dogs actively accumulated 2,4,5-T. Accumulation was oxygen dependent and saturable, and as reduced in the presence of other anions (*p*-aminohippurate and probenecid). A reduction in the potassium concentration of the medium reduced accumulation of 2,4,5-T by rat tissue but not by dog tissue. Acetate in the medium increased accumulation of the herbicide in dog but not in rat tissue. Finally, the ability of renal tissue from newborn rats to accumulate 2,4,5-T was significantly less than that of adult tissue. It is concluded that the primary route of renal elimination of 2,4,5-T is active secretion of the compound. The greater ability of adult rat tissue to transport PAH explains the shorter biological half-life of 2,4,5-T in this species. (Author abstract by permission)

75-1362. Eisenbrand, G.; Ungerer, O.; Preussmann, R. (Inst. Toxikol. Chem. Dtsch. Krebsforschungszentr., Heidelberg, Germany). **Rapid formation of carcinogenic *N*-nitrosamines by interaction of nitrite with fungicides derived from dithiocarbamic acid in vitro under simulated gastric conditions and in vivo in the rat stomach.** *Food Cosmet. Toxicol. 12(2): 229-232; 1974.* (12 references)

The formation of the carcinogen dimethylnitrosamine (DMN) from the fungicide bis-(dimethyldithiocarbamato)zinc (ziram) in the presence of sodium nitrite has been studied in vitro under conditions similar to those existing in the stomach and also in vivo in the rat. Under the selected conditions, the optimum pH for the formation of DMN was 1.5-2.0. At pH 2.0, more than 1 mg DMN was produced after a 10-min incubation of 10^{-4} moles ziram with a twentyfold molar excess of nitrite. This corresponds to about 8% of the theoretical yield, assuming that two molecules of the carcinogen were formed from one molecule of ziram. Tris(dimethyldithiocarbamato)iron (ferbam) and bis-(1-pyrrolidinylthiocarbonyl)disulfide (dipyrrolidylthiuramdisulfide; DPTD) produced DMN and *N*-nitrosopyrrolidine respectively under the same conditions. (Author abstract by permission)

75-1363. Vitorovic, S.; Kapor, S.; Neskovic, N.; Zezelj, A. (Department of Pesticides INEP, Belgrade-Zemun, Yugoslavia). **Sadrzaj parationa u tkivima akutno otrovanih pacova. [Parathion content in individual tissues of acutely poisoned rats.]** *Arh. Hig. Rada Toksikol. 22(2): 151-154; 1971.* (8 references) (Serbo-Croatian)

Parathion content was determined using gas-liquid chromatography in the brain, liver, and muscle tissues of rats poisoned by a single intraperitoneal injection. A chromatograph equipped with a phosphorus detector was used. The column was packed with a mixture of two stationary phases (silicone oil DC-200 and QF-1) on Chromosorb W-HMDS. No measurable quantities of parathion were found in the brain and during the first 60 minutes after the injection there was no parathion in the liver or abdominal muscles either. Over longer time intervals the quantities of parathion detected were in the range of 0.125 to 7.158 mg/g of tissue.

75-1364. Wilhelm, K.; Plestina, R.; Svetlicic, B. (Inst. Med. Res. Occup. Health, Zagreb, Yugoslavia). **Toksicnost ekatina i njegovo djelovanje na profesionalno eksponirane radnike. [The toxicity of Ekatin and its action on occupationally exposed workers.]** *Arh. Hig. Rada Toksikol. 25(2): 319-320; 1974.* (Serbo-Croatian)

Insecticides from the organophosphorus group are still frequently used in the fight against pests in agriculture, forestry, public health, and veterinary science. Inhibitory properties of thiometon [S-2-(ethylthio)ethyl dimethyl phosphorodithioate], active component of the organophosphorus insecticide Ekatin, were studied in vitro on human blood cholinesterase. Its toxic effects were studied on laboratory animals. Results of the control check-up of workers employed in the production of Ekatin are also presented. The inhibitory effect of thiometon was more pronounced towards plasma cholinesterase ($I50 = 2.8 \times 10^{-4}$ M) than towards erythrocyte cholinesterase ($I50 = 4.0 \times 10^{-4}$ M). Acute oral toxicity tests in rats showed that the concentrate used in the production of Ekatin was more toxic (LD50 = 74.9 mg/kg) than was expected from the data obtained on the toxicity of the active matter (LD50 = 130 mg/kg). Lowered cholinesterase activity level was found in the blood of several workers, but apart from transient subjective complaints no noticeable noxious effects were recorded.

75-1365. Miyao, N.; Ishiguro, S.; Kono, I.; Yasuda, N. (Lab. Vet. Pharmacol., Kagoshima Univ. Kagoshima, Japan). **[Studies on the toxicity of carbamate compounds. I. Subchronic toxicity of 3-methyl-5-isopropylphenyl-*N*-methyl carbamate.]** *Bull. Fac. Agric. Kagoshima Univ. 22: 131-144; 1972.* (13 references) (Japanese)

Eighty rats and mice were divided into 4 groups and 3-methyl-5-isopropylphenyl-*N*-methylcarbamate (promecarb) was administered orally each day for a test period of 3 months. Drug levels given were 50 mg/kg to the rats and 20 mg/kg to the mice in group I, 33 and 13.7 mg/kg in group II, 22 and 8.9 mg/kg in group III, and group IV were controls. The acute oral LD50 was 60 (42.3-85.1) mg/kg in the rat and 39.5 (33.1-47.2) mg/kg in the mouse. Deaths to date include 5 male and 8 female rats and 8 male and 10 female mice in group I; 2

male and 5 female rats and 3 male and 8 female mice in group II; 1 male rat and 2 male and 2 female mice in group III; and 1 male rat and 1 male and 1 female mouse in group IV. A slight but irregular suppression of growth was observed in the rats but not in the mice. No drug-induced hematologic changes were noted. Although almost no difference in the levels of plasma GOT, GPT, urea nitrogen, and protein was noted between treated and control animals, blood sugar levels of treated animals were high. Plasma and brain cholinesterase activity was totally suppressed in treated animals. Organ weights hardly changed between control and treated groups, but the spleen and ovary weights were significantly reduced. The principal histopathologic changes recognized in animals which died or were killed after three months included hepatitis; liver degeneration; peribronchitis; hyperemia of liver, kidney, lung, and adrenals; myocardial necrosis; and cerebral hemorrhage.

75-1366. Miyao, N.; Ishiguro, S.; Yasuda, N. (Lab. Vet. Pharmacol., Kagoshima Univ., Japan). **[Studies of the toxicity of carbamate compounds. II. Subchronic toxicity of *m*-(((dimethylamino)methylene)amino) phenylmethylcarbamate.]** *Bull. Fac. Agric. Kagoshima Univ. 22: 145-153; 1972.* (3 references) (Japanese)

Eighty rats were divided into 4 groups and orally administered *m*-(((dimethylamino)-methylene)amino)-phenylmethylcarbamate (formetanate) daily throughout a test period of 3 months. Levels of the drug were 200 mg/kg in group I, 10 mg/kg in group II, 5 mg/kg in group III, and 0 mg/kg in group IV. In rats of groups I and II, whole body tremors and suppression of spontaneous movement (though ceasing after about 20 to 40 minutes) were observed about 10 minutes after administration. To date 5 males and 2 females in group I, 3 males and 2 females in group II, 3 males and 1 female in group III, and 1 male in group IV have died. No growth suppression was observed during the test periods. Almost no change was observed in erythrocyte and leukocyte counts, hemoglobin concentration, hematocrit value, and differential leukocyte count in the treated animals. Although the levels of plasma GOT, GPT, urea nitrogen, and protein hardly changed between control and treated animals the plasma and brain cholinesterase activities were ascertained to be slightly less in treated than control rats. Organ weights in treated animals were observed lower (male thyroid gland, adrenal, and testis) or higher (male liver and spleen, and female lung and ovary) than in the control group. In animals which died in the course of studies or were sacrificed after 3 months, histopathologic changes observed frequently were hepatitis, liver degeneration, nephritis, and peribronchitis. Of these changes, liver and kidney degeneration were considered to be due to the effect of the drug; interstitial edema of the testis, disappearance of colloid in thyroid gland, and heart muscle necrosis were of doubtful cause.

75-1367. Starek, A. (Author address not given). **Doswiadczemia nad wchlanianiem z przewodu pakarmowego szczurow izomeru gamma szesciochlorocykloheksanu (Lindanu). [Experiments on absorption of γ-hexachlorocyclohexane isomer (lindane) from alimentary tract in rats.]** *Folia Med. Crakov. 14(2): 235-252; 1972.* (17 references) (Polish)

Results are presented on the absorption of lipid-soluble poisons from the alimentary tract, and absorption of γ-BHC (lindane) in the presence of non-laxative doses of olive oil and castor oil, and laxative doses of castor oil. Experiments were carried out with 181 male Wistar rats weight about 230 g. The animals were divided into five groups, in which substances were introduced operatively into the small intestine. The control group received lindane in aqueous suspension in dose of 200 mg/kg body weight. Three groups of rats received, in addition, 1.0 ml of olive oil and 0.5 ml of castor oil (non-laxative doses). In the fifth group only water was introduced into the intestine. Each experimental group was divided into four subgroups according to the duration of the experiment (1, 4, 6, and 12 hours) counting from the moment of introduction of the studied substances to autopsy of the animals. Observations were made on amount of toxic substance absorbed from the intestine, retention of lindane in the parenchymal organs, changes in guanase and diphosphate aldolase activities, changes in serum protein levels and its fractions, and mortality of the rats. The greatest absorption of lindane, greatest retention in the parenchymal organs, highest activity of the studied enzymes, changes in the serum protein fractions, and highest mortality of the rats were observed in the control group of rats. In the group of rats treated with lindane and olive oil, resorption of the compound and biochemical changes were less pronounced than in the control group. Retention of the compound in parenchymal organs and mortality were the same as in the first group of rats. Smallest absorption of lindane and its smallest retention in parenchymal organs, as well as least pronounced biochemical changes, were observed in rats treated with lindane and castor oil in non-laxative doses. In the group of rats treated with lindane and castor oil in laxative doses, absorption of the poison was enhanced, and biochemical changes increased in the course of the experiment. The experiments showed that olive oil slightly diminishes absorption of lindane, and that the inhibitory effect of castor oil depends on its dosage – distinct after non-laxative doses, and very slight after laxative doses.

75-1368. Shchitskova, A. P. (F. F. Erisman Moscow Sci. Res. Inst. Hyg., Moscow, USSR). **Nekotoryye aspekty otsenki spetsificheskogo deystviya na organizm pestitsidov. [Some aspects of the assessment of the specific action of pesticides on the organism.]** *Gig. Sanit. 39(8): 76-78; 1974.* (Russian)

Studies on the specific effects of pesticides on the human and animal organism are reviewed. Certain pesti-

cides, especially zineb, cause changes in the reactivity of the organism and bring about allergic reactions. Zineb, applied in a dose of 1/5,000 LD50, was found to cause changes in the immunological reactivity of the organism for a long time. Ziram and zineb caused considerable disturbances in the spermatogenesis and oogenesis in rats, more or less complete loss of fertility in both sexes, and increased postnatal and embryonal lethality, and had a mutagenic effect. Zineb and trichlorfon increased the frequency of chromosome aberrations and dominant lethal mutations in mice. The early stage of spermatocytes was found to be most susceptible to the mutagenic effect of trichlorfon in coincidence with the major part of the DNA synthesis.

75-1369. Petre, I.; Calciu, A. (Inst. Ig. Sanat. Pub., Bucharest, Romania). **Studiul experimental al unor pesticide cu mare persistenta in apa. [An experimental study of pesticides which display higher persistence in water.]** *Igiena 22(2): 83-92; 1973.* (18 references) (Romanian)

Lindane, the gamma-isomer of BHC, and Dibutox, dinitro-*sec*-butylphenol (DNBP), were employed in a chronic experiment with white rats. Daily amounts administered were 1 mg/kg of body weight. The body weight of the animals increased after administration of Dibutox and was lowered by the administration of lindane. Hepatic weight was also increased by the administration of lindane and a stimulating effect was found on the activity of some enzymes, such as succinate dehydrogenase.

75-1370. Cenci, P.; Cavazzini, G. (Inst. Ig., Univ. Ferrara, Ferrara, Italy). **Effetti della monofluoroacetammide (MNFA) sulla microflora dell'ambiente. Nota II. Effetti della MNFA sul ciclo del Carbonio. Effects of monofluoroacetamide (MNFA) on environmental microflora. II. Effects of MNFA on the carbon cycle.]** *Ig. Mod. 65(9-10): 476-496; 1973.* (32 references) (Italian)

The effects of MNFA on microorganisms involved in the natural carbon cycle are reported. Using concentrations of 1, 10, 100, 500 and 100 γ/ml of MNFA, microorganisms were tested for: cellulolysis (*Cellvibrio viridis*), growth and lactic acid production (*Lactobacillus bulgaricus*), growth and propionic acid production (*Propionibacterium shermanii*), growth and ethyl alcohol production (*Saccharomyces cerevisiae* var. *ellipsoideus*), growth and acetic acid production (*Acetobacter aceti*), growth and amylolysis (*Aspergillus oryzae*), growth and citric acid production (*Aspergillus niger*), and growth of *Streptomyces* sp., *Penicillus* sp. and *Sclerotinia fructicola*. It is shown that MNFA inhibits two enzymatic reactions, both catalyzed by NADH (i.e., ethyl alcohol and lactic acid production); however, citric acid production seems to be stimulated. The latter result supports the hypothesis that MNFA may inhibit aconitase as well as isocitric dehydrogenase, taking into consideration the possible deviation of the Krebs cycle through the glyoxalate pathway.

75-1371. Andia, A. M. G.; Street, J. C.* (Dep. Chem. Biochem., Utah State Univ., Logan, UT 84322). **Dietary induction of hepatic microsomal enzymes by thermally oxidized fats.** *J. Agric. Food Chem. 23(2): 173-177; 1975.* (26 references)

Functional changes associated with the hepatomegaly commonly observed upon feeding thermally oxidized (TO) fats were investigated. Rats were fed purified diets in which the fat consisted of fresh corn oil, TO oil, or the proportional amount of non-urea-adduct-forming material (NUAF) from TO oil plus fresh oil. Increases in relative liver weights and the concentrations of microsomal protein and endogenous malondialdehyde were observed when TO oil or NUAF plus fresh oil were fed rather than pure fresh oil with two types of dietary protein, casein and soy. Both the basal and DDT-induced mixed function oxidase activities were higher in animals fed TO oil and NUAF than in those given fresh oil. The TO oil also increased cytochrome P-450 and the activity of *S*-adenosylmethionine:phosphatidylethanolamine methyltransferase whereas the NUAF did not. Oxidized fat thus appears to stimulate SER proliferation and induce a complex of microsomal enzymes. (Author abstract by permission)

75-1372. Kulkarni, A. P.; Mailman, R. B.; Hodgson, E.* (Dep. Entomol., North Carolina State Univ., Raleigh, NC 27607). **Cytochrome P-450 optical difference spectra of insecticides. A comparative study.** *J. Agric. Food Chem. 23(2): 177-183; 1975.* (36 references)

The cytochrome P-450 optical difference spectra of insecticides were examined using hepatic microsomes from sheep, rabbit, rat, and mouse as well as abdominal microsomes from insecticide-resistant (Fc) and insecticide-susceptible (CSMA) house flies. The results generally conform to the hypothesis that type II binding spectra are elicited by compounds containing a nitrogen atom with a sterically accessible pair of nonbonded electrons and, in addition, certain compounds with nucleophilic oxygen atoms. Other compounds generally exhibit type I spectral binding or do not form detectable spectra. Exceptions include certain pyrethroids which, while giving typical type I difference spectra with mammalian microsomes, gave rise to an unusual spectrum in insects, one with a peak at 415–418 nm and trough at 445–447 nm. Rotenone also gave rise to unusual difference spectra with hepatic microsomes of sheep and rabbit. Differences in the ratios of spectral size between species indicate that qualitative differences in the cytochrome P-450 of different species are common. (Author abstract by permission)

75-1373. Lin, T. H.; North, H. H.; Menzer, R. E.* (Dep. Entomol., Univ. Maryland, College Park, MD 20742).

Metabolism of carbaryl (1-napthyl *N*-methylcarbamate) in human embryonic lung cell cultures. *J. Agric. Food Chem. 23(2): 253-256; 1975.* (13 references)

Carbaryl (1-naphthyl *N*-methylcarbamate) was metabolized oxidatively by primary human embryonic lung cells in culture. After incubation of [^{14}C] carbaryl (300,000-400,000 dpm, about 3 μg) for 72 hr, 70% of the recovered radioactivity resulted from oxidative metabolites, with 30% being water soluble. The total recovery of added radioactivity was over 99%. Protein and nucleic acids determinations on cellular materials revealed that carbaryl at the applied dosage level did not inhibit cell growth and enzyme syntheses. Unconjugated metabolites from both cells and cell sonicates were identified as 1-naphthol, 5-hydroxycarbaryl, 4-hydroxycarbaryl, and 5,6-dihydro-5,6-dihydroxycarbaryl. The water-soluble materials released after HCl hydrolysis were identified as 4-hydroxycarbaryl, 1,4-naphthalenediol, and 5,6-dihydro-5,6-dihydroxycarbaryl. Several unknowns remained to be identified. The conjugated metabolites were possibly not *O*-glucuronides because β-glucuronidase treatment did not free aglycones from conjugation. Furthermore, the addition of UDPGA and other cofactors to the cell sonicates did not effect any increase in conjugations. (Author abstract by permission)

75-1374. Lin, T. H.; North, H. H.; Menzer, R. E.* (Dep. Entomol., Univ. Maryland, College Park, MD 20742). **The metabolic fate of chlordimeform [*N*-(4-chloro-*o*-tolyl)-*N'*,*N'*-dimethylformamidine] in human embryonic lung cell cultures.** *J. Agric. Food Chem. 23(2): 257-258; 1975.* (8 references)

The metabolism of [^{14}C] chlordimeform (*N*-(4-chloro-*o*-tolyl)-*N'*,*N'*-dimethylformamidine) by primary human embryonic lung cells in culture was studied. Chlordimeform was quite susceptible to degradation by the cell cultures. Over 97% of the applied chlordimeform was changed by the cells after 72 hr of incubation, with the major metabolites appearing as *N*-formyl-4-chloro-*o*-toluidine (81.9%) and 4-chloro-*o*-toluidine (2.3%). Other minor metabolites of chlordimeform included *N*-demethylchlordimeform (*N*-(4-chloro-*o*-tolyl)-*M'*-methylformamidine) and two unknown metabolites. When [^{14}C] chlordimeform was incubated in medium without cells for 72 hr, the compound was decomposed to *N*-formyl-4-chloro-*o*-toluidine (10%) with the remainder being unchanged. Water-soluble metabolites of chlordimeform were formed in insignificant quantities in both controls and test flasks.

75-1375. Mehendale, H. M.; Fields, M.; Matthews, H. B. (Natl. Inst. Environ. Health Sci., Research Triangle Park, NC 27709). **Metabolism and effects of hexachlorobenzene on hepatic microsomal enzymes in the rat.** *J. Agric. Food Chem. 23(2): 261-265; 1975.* (30 references)

Seven days following a single oral dose of hexachlorobenzene (HCB-^{14}C) to adult male rats, approximately 16% of the dose was excreted in the feces and less than 1% in urine. Metabolites of HCB included pentachlorobenzene, tetrachlorobenzene, pentachlorophenol, and four unknowns; all were abstracted from urine and none was detected in the feces. Seventy per cent of the total dose remained in the body 7 days after administration, fat being the major depot. This residue consisted mainly of HCB with traces of the dechlorinated metabolites. Reductive dechlorination of HCB was catalyzed by an enzyme located in the microsomal fraction of liver, lung, kidney, and intestine. Added NADPH was required for the formation of pentachlorophenol. Hepatic glucuronyl transferase, aniline hydroxylase, ethylmorphine and *p*-nitroanisole demethylases, and cytochromes P-450 and b_5 were induced by HCB pretreatment. Urinary coproporphyrin levels more than doubled by the fourth day of treatment and remained relatively constant thereafter, although apparent signs of porphyria were not observed in these animals. (Author abstract by permission)

75-1376. Fries, G. F.; Marrow, G. S. (U.S. Dep. Agric., Agric. Res. Serv., Agric. Environ. Qual. Inst., Beltsville, MD 20705). **Retention and excretion of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin by rats.** *J. Agric. Food Chem. 23(2): 265-269; 1975.* (10 references)

Rats were fed 7 or 20 ppb of 2,3,7,8-[^{14}C] tetrachlorodibenzo-*p*-dioxin (TCDD) in their diets for 42 days. Dose-related effects on feed consumption and growth were more severe in males than in females. Both levels of TCDD significantly increased liver weights, but 7 ppb caused the greater increase. Total retention of TCDD was closely related to total intake at any given time period. Males and females did not differ significantly in total retention, but 85% of the TCDD was in the liver of males, whereas only 70% was in the liver of females. Total retention was 5.5, 7.5, and 10.0 times daily intake at 14, 28, and 42 days, respectively. Kinetic analysis indicated that at steady state, total retention would be approximately 10.5 times the average daily intake. When feeding stopped, TCDD residues were eliminated from the body with half-lives of 12 and 15 days for males and females, respectively. (Author abstract by permission)

75-1377. Giurgis, G. N.; Brindley, W. A.* (Dep. Biol., Utah State Agric. Exp. Stn., Utah State Univ., Logan, UT 84322.) **Carbaryl penetration into and metabolism by alfalfa leafcutting bees, *Megachile pacifica*.** *J. Agric. Food Chem. 23(2): 274-279; 1975.* (14 references)

Carbaryl metabolism by and penetration into adult alfalfa leafcutting bees, *Megachile pacifica* (Panzer) (= *rotundata* Fabr.), were studied in relation to sex, age, and exposure to piperonyl butoxide. Unchanged carbaryl was a principal compound isolated from the bees after 4 hr, being more prominent in 4-day-old males (48% of extractable metabolites) and less prominent in

4-day-old females (22%) and 1-day-old males (26%). 1-Naphthol, 5,6-dihydro-5,6-dihydroxy-carbaryl, 5-hydroxycarbaryl, and carbaryl were recovered from acid hydrolysis of water-soluble conjugates. Two minor organosoluble metabolites were not identified as were none of the conjugating moieties. Carbaryl persistence in the bees was closely related to carbaryl susceptibility. The results demonstrated that the differences in carbaryl LD50 values with and without piperonyl butoxide treatment are more meaningful than the quotient of these values. (Author abstract by permission)

75-1378. Suzuki, T.; Uchiyama, M. (Pharm. Inst., Tohoku Univ., Sendai, Japan). **Pathway of nitro reduction of parathion by spinach homogenate.** *J. Agric. Food Chem. 23(2): 281-286; 1975.* (12 references)

The nitro reduction of parathion to aminoparathion by spinach homogenate under anaerobic conditions proceeds via hydroxylaminoparathion as intermediate metabolite. Hydroxylaminoparathion was stable in the acidified reaction medium and therefore detectable by thin-layer chromatography. However, in the alkaline medium, it underwent autoxidation to nitrosoparathion, which was detectable by both thin-layer chromatography and gas chromatography. Nitrosoparathion itself was not demonstrated as an intermediate metabolite, but the one added to the subject reaction system was easily reduced to aminoparathion via hydroxylaminoparathion. It was also converted rapidly to hydroxylaminoparathion by NADPH. These results suggest the possibility that nitrosoparathion may act as one of two consecutive intermediate metabolites. (Author abstract by permission)

75-1379. Dubey, H. D.; Riera, A.; Rodriguez, R. L. (Agric. Exp. Stn., Univ. Puerto Rico, Mayaguez Campus, Rio Piedras, PR). **Effect of the nematocides nemagon and DD on mineralization, nitrification, soil microbial population, and soil fertility status of two tropical soils.** *J. Agric. Univ. P. R. 59(1): 43-50; 1975.* (6 references)

The effect of the nematocides dibromochloropropane and DD on the soil microbial population in two Puerto Rican soil types was investigated. DD was extremely toxic to the nitrifying bacteria, much more so than dibromochloropropane. It inhibited ammonium ion oxidizing bacteria but not *Nitrobacter*. DD eliminated all types of microbes in every soil when applied at the 2000 ppm level. This effect was more drastic in acid than alkaline soils. At the 200 ppm rate the bacterial population increased, indicating that bacteria may be using this chemical in their metabolism, and are thus instrumental in its detoxification. Bacteria were suppressed at rates higher than 200 ppm. Actinomycetes and fungi were suppressed drastically.

75-1380. Galea, V.; Colosi-Esca, D.; Lucuta, V. (Inst. San. Publ. Cercet. Med., Cluj, Romania). **Dinamica toxicitatii carbetoxului si fosfotoxului R. [Dynamics of the toxicity of carbetox and of fosfotor R.]** *Igiena 23(2): 87-96; 1974.* (23 references) (Romanian)

One-seventh LD50 of Carbetox (malathion 37%) and Fosfotox R (dimethoate 35%) were administered daily for three weeks to the peritoneal cavity and the functional, biochemical and anatomical changes reported. These changes were followed for a further 3 weeks after cessation of the drugs. Investigations concerned general indicators of toxicity (death rate, body weight, histologic appearance and organ weight, spontaneous motility) as well as indicators related to the pecularities of the active substances (cholinesterase activity inhibition, hepatic tissue transaminases, respiration). Results (especially those concerning the variations in weight, spontaneous motility, and cholinesterase activity) indicate that, in conditions of exposure to low and moderate doses, Fosfotox R is a pesticide with a lower toxicologic risk than Carbetox and has optimal possibilities for adaptation by mammals.

75-1381. Araki, M.; Mizuhira, V. (Tokyo Med. Dent. Univ., Tokyo, Japan). **Degenerative changes of the ciliary body of a chronically phosphorism-inflicted beagle.** *J. Electron Microsc. 22(3): 287; 1973.*

Ethylthiometon (10 mg, p.o.) was administered daily to a six-months old pure beagle for a 2-yr period, for a total of 4600 mg. The basement membrane between the ciliary muscular cells was extremely thickened and electronic density was increased, presumably because of bismuth. Degeneration of mitochondria in the muscular cells, decrease of myofilament, disappearance of ribosomes, and decrease of nuclear chromatin were observed. Phosphorus and especially sulfur were readily detected in the thickened basement membrane and cell nucleus by the process of the non-dispersive type of electron probe X-ray microanalyzer (EDAX) fitted on an electron microscope.

75-1382. Armstrong, D. A.; Millemann, R. E. (Dep. Fish. Wildl., Mar. Sci. Cent., Oregon State Univ., Newport, OR 97365). **Effects of the insecticide carbaryl on clams and some other intertidal mud flat animals.** *J. Fish. Res. Bd. Can. 31(4): 466-470; 1974.* (13 references)

The insecticide carbaryl (Sevin) significantly reduced numbers of juvenile clams in plots treated with 2.3 and 4.6 kg (5 and 10 lb/acre). Pooling of samples taken at 1, 2, 4, 15, and 30 days after treatment showed mean clam numbers per m^2 in untreated and treated plots were 364, 283, and 224, respectively, the reductions from the controls being 22 and 38%. Clam species differed in susceptibility to carbaryl; for example, numbers of the gaper clam (*Tresus capax*) were reduced by 58 and 69% at the low and high application rates in relation to those from the control plots, and the bent-nosed clam (*Macoma nasuta*) by 9 and 28%. There was

no reduction in numbers of polychaete and nemertean worms. A carbaryl application of 2.3 kg/acre was as effective in controlling ghost shrimp (*Callianassa californiensis*), an oyster pest, as 4.6 kg/acre. Signs of stress and numbers affected are given for other mud flat animals. (Author abstract by permission)

75-1383. Meehan, W. R.; Norris, L. A.; Sears, H. S. (U.S. For. Serv., For. Sci. Lab., Juneau, AK 99801). **Toxicity of various formulations of 2,4-D to salmonids in southeast Alaska.** *J. Fish. Res. Bd. Can. 31(4): 480-485; 1974.* (17 references)

To determine acute toxicity to juvenile (1) pink, chum, coho, and sockeye salmon, (2) Dolly Varden char, and (3) rainbow trout, 2,4-D acid, butyl and isooctyl esters were tested in southeast Alaska. A comparable test was made in Oregon using coho salmon fingerlings. The mean percent mortality after 96 hr and the highest concentration of herbicide that did not produce any mortality were determined for each formulation tested. At less than 50 ppm 2,4-D acid produced no mortality except in pink salmon fry. The butyl ester was most toxic causing nearly complete mortality in all species at concentrations > 1.0 ppm and the isooctyl ester least toxic of the ester formulations. Alaskan and Oregon coho fingerlings were similar in their responses to 2,4-D acid, butyl, and isooctyl esters. The toxicities of three different formulations of isooctyl ester, a PGBE ester, and butyl ester to Alaskan coho fingerlings were also determined. There were few or no differences in toxicity among isooctyl ester formulations. The butyl and PGBE esters were similar in toxicity. (Author abstract by permission)

75-1384. Pruzandky, J. J.; Patterson, R. (Dep. Med., Northwestern Univ. Med. Sch., Chicago, IL.). **The diisopropylfluorophosphate inhibitable step in antigen-induced histamine release from human leukocytes.** *J. Immunol. 114(3): 939-943; 1975.* (13 references)

The effects of time and temperature on DFP inhibition of histamine release from human leukocytes were investigated. The temporal characteristics of DFP activity are compared with those of ethylenediaminetetraacetate and 2-deoxyglucose. When DFP was added to cell suspensions at various times after incubation with antigen, it was never an effective inhibitor if added after 4 min, while ethylenediaminetetraacetate and 2-deoxyglucose still inhibit the reactions when added 5 min after antigen. Leukocytes incubated for 90-120 min at 0°C react with specific antigen since they subsequently release significant quantities of histamine after washing and reincubation at 37°C without addition of antigen. Such priming at 0°C is at least equivalent to priming for 2-4 min at 37°C. During antigen priming at 0°C, the cells are not activated beyond the step in the release sequence which is inhibited by DFP. Incubation of lekuocytes with specific antigen at 37°C for 3 min resulted in significant but not quite complete priming for subsequent histamine release in the absence of antigen. After 5 min. incubation with antigen at 37°C almost all leukocytes have progressed beyond the stage which is inhibited by DFP.

75-1385. Friend, M.; Trainer, D. O. (U.S. Fish Wildl. Serv., Univ., Wisconsin, Madison, WI 53706). **Experimental DDT-duck hepatitis virus interaction studies.** *J. Wildl. Manage. 38(4): 887-895; 1974.* (12 references)

In three experiments, mallards (*Anas platyrhynchos*) were fed two levels of DDT, then given challenge inoculations of two levels of duck hepatitis virus (DHV). In experiments I (20-day-old birds) and II (29-day-old birds), mortality occurred over a longer period and gross liver lesions were less common and less severe among birds given both DDT and DHV than among DHV controls. In experiment II, mortality ranged from 6 percent (DHV control) to a significantly higher ($P < 0.01$) 47 percent (900 ppm DDT plus DHV) at a challenge dose of $10^{0.5}$ duck LD50 of DHV. DDT residues in the brains of 29-day-old ducklings given both DDT and DHV were greater in birds that died than in survivors (about 80 to 200 percent greater in birds given 500 ppm DDT, 250 to 255 percent greater in birds given 900 ppm DDT). Only one death (apparently from DDT) occurred among the mature male mallards used in experiment III, but brain and whole-body DDT residues in mallards killed the day after DHV challenge were much higher than in those killed before challenge. These results indicate that exposure to sublethal levels of DDT altered the response of mallards to DHV. (Author abstract by permission)

75-1386. Friend, M.; Trainer, D. O. (U.S. Fish Wildl. Serv., Univ. Wisconsin, Madison, WI 53706). **Experimental dieldrin-duck hepatitis virus interaction studies.** *J. Wildl. Manage. 38(4): 896-902; 1974.* (15 references)

In three experiments, mallards (*Anas platyrhynchos*) were fed two levels of dieldrin, then given challenge inoculations of two levels of duck hepatitis virus (DHV). In experiments I (20-day-old birds) and II (29-day-old birds), mortality occurred over a longer period and gross liver lesions were less common and less severe among birds given both dieldrin and DHV than among DHV controls. In experiment II, mortality ranged from 6 percent (DHV control) to a significantly higher ($P < 0.01$) 57 percent (40 ppm dieldrin plus DHV) at a challenge dose of $10^{0.5}$ duck LD50 of DHV. Dieldrin residues in the brains of ducklings given both dieldrin and DHV were greater in birds that died than in survivors (about 30-150 percent greater in birds given 20 ppm dieldrin, 170-1,410 percent greater in birds given 40 ppm dieldrin). Only one death (apparently due to dieldrin) occurred among the mature male mallards used in experiment III. These results indicate that exposure to sublethal levels of dieldrin altered the response of mallards to DHV. (Author abstract by permission)

75-1387. Messick, J. P.; Bizeau, E. G.; Benson, W. W.; Mullins, W. H. (Dep. Zool., Univ. British Columbia, Vancouver, British Columbia, Canada). **Aerial pesticide applications and ring-necked pheasants.** *J. Wildl. Manage. 38(4): 679-685; 1974.* (21 references)

In 1970 aerial applications were made to a 24-hectare section in southwestern Idaho utilizing ethyl parathion in May, demeton and DDT-toxaphene in June, and trichlorfon and demeton in July. Aerial application results in a very uneven distribution of chemicals on the ground. Gas chromatographic analysis of residues collected on glass plates during aerial applications showed ranges of 0–2058 μg per plate within the treated area. Observations on penned and radio-equipped wild pheasants (*Phasianus colchicus*) revealed no direct mortality from the spraying. Penned pheasants 5–15 days old exhibited symptoms of organophosphorus poisoning, but recovered. Cholinesterase and weight gains did not differ signficantly in treated and untreated penned pheasants. According to the results of sweep net samples, insect populations were significantly lower in the treated area compared to the untreated area. Juvenile pheasants collected in the treated area consumed significantly fewer insects than birds taken in the untreated area. Applications of certain organochlorine and organophosphorous pesticides probably kill some pheasants, especially young birds, but results suggest that use of demeton, ethyl parathion, DDT-toxaphene, and trichlorfon at recommended application rates does not cause major pheasant losses in southwestern Idaho. (Author abstract by permission)

75-1388. Ogawa, E.; Suzuki, S.; Tsuzuki, H.; Kawajiri, M. (Sch. Pharmacol., Fac. Med., Gumma Univ., Maebashi, Gumma, Japan). **[An experimental study on distribution and excretion of ethyl mercury chloride.]** *Kitakanto Igaku (Kitakanto Med. J.) 24(4): 229-235; 1974.* (Japanese)

Large amounts of ethylmercury compounds have been used although they were banned. Radiolabeled (^{203}Hg) ethylmercury chloride suspended in olive oil was administered p.o. to ddN male and Wistar male and female rats. The absorption, retention, excretion, and distribution of radioactivity were traced, as well as the accelerating effect on excretion by addition of several drugs. Findings revealed biological half lives of 4.4 days in 10 wk old male mice; 10.1, 9.3, and 8.7 days in 6, 8, and 10.5 wk old male rats, respectively; and 7.0 days in 10.5 wk old female rats. Mercury levels decreased in the order kidney, blood, liver, pancreas, spleen, and brain 1 hr after administration. Drugs found effective in accelerating Hg excretion were: DL-penicillamine, 2-mercaptopropionyllysine, and large amounts of glutathione.

75-1389. Knauf, W.; Schulze, E. F. (Farbwerke Hoechst AG, Frankfurt am Main, Germany). **New findings on the toxicity of endosulfan and its metabolites to aquatic organisms.** *Meded. Fac. Landbouwwetensch. Rijksuniv. Gent 38(3): 717-732; 1974.* (9 references)

The toxicity of endosulfan and its metabolites to aquatic organisms was investigated. The LC50 (48 hr) was determined for α-endosulfan β-endosulfan, endosulfan 35 EC, endosulfan sulfate, endosulfan alcohol, endosulfan ether, endosulfan hydroxyether, and endosulfan lactone to test organisms from the group Pisces, Crustacea, Insecta, Mollusca, and Oligochaeta and to the alga *Chlorella vulgaris*. For all invertebrates the LC50 is 1-10 ppm. When sulfur was removed from the molecule, the effect on fish was lost. The first impact of endosulfan on *C. vulgaris* was observed in concentrations greater than 2 ppm (reproduction). After 5 days of exposure this effect is fully reversible. At concentrations of 0.01 ppm, there was no effect on the reproduction rate, photosynthesis, and biomass production of this alga. The LD50 values span a range of 10^4 for the various animal species tested.

75-1390. Giermaziak, H. (Dep. Gen. Exp. Pathol., Med Acad., Lodz, Poland). **Badania nad wplywem zwiazkow fosfoorganiczynch na ustroj na modelu Intrationu. [Studies on the effect of organophosphorus compounds on the body as exemplified by "Intration."]** *Med. Pracy 24(5): 487-494; 1973.* (Polish)

Intration (thiometon) was administered i.p. at 100 mg/kg to gray rabbits (2-3 kg), causing acute intoxication. Acid-kathepsin protease (KT), beta-glucuronidase (β-GRD), acid and alkaline phosphatase (AcP and AP), and cholinesterase (AChE and ChE) were determined in serum and white blood cell fractions (supernatant, homogenate, and lysosomal fraction) before and 24 hours after poisoning. Determinations were also made after treatment of poisoned animals with pralidoxime (i.v. 7 mg/kg) or atropine sulfate (i.m. 0.0005/animal). In addition RBC, hemoglobin color index, WBC, and blood glucose levels were determined, and electrocardiograms and histopathologic examinations of heart muscle performed. Results were analyzed statistically. A significant increase in the activity of acid hydrolases and alkaline phosphatase and decrease in cholinesterases was found in the serum. The activity of β-GRD and AcP was increased in all fractions of white blood cells, and activity of KT and AChE + ChE mainly in the homogenate. There was a marked increase in WBC and decrease in RBC and blood glucose. The ECG showed a reduced heart rate, lengthened PQ interval and flattening of T segments. The myocardium showed hyperemia and foci of inflammatory, steatotic, degenerative, and necrotic changes. After administration of PAM (pralidoxime) the enzymatic activities returned to normal except for AP in serum and AcP and AP in supernatant and homogenate fractions where it remained elevated. There was also a normalization of measured parameters in peripheral blood and ECG except for heart rate which remained slow. Atropine sulphate had no effect on heart rate or T segment, hemoglobin or blood

glucose. Although both compounds had a beneficial effect on the PQ interval neither could prevent long term damage to the heart muscle.

75-1391. Galasinska-Pomykol, I.; Stefanska-Sulik, E. (Dep. Anat. Inst. Biol. Morphol., Acad. Med., Bialystok, Poland). **Analiza morfologiczna komorek jadra niskowzgorzowego swinek morskich poddanych dzialaniu pestycydu "Alvit-55". [Morphological analysis of cells of the subthalamic nucleus of guinea pigs treated with the pesticide "Alvit-55."]** *Med. Pracy 25(1): 27-37; 1974.* (18 references) (Polish)

"Alvit-55" whose main component is dieldrin was administered at 25 mg/kg to female guinea pigs (500-650 g). Convulsions occurred in some animals (group I) but not in others (group II). Five animals of each group were sacrificed 4 hours after administration of the compound. Cells of the subthalamic nucleus were isolated and a detailed morphological analysis of 3,000 (200 from each animal) cell nuclei from different types of neurons made. The volume and shape of the nucleus and location of the nucleolus were determined in multipolar, fusiform, oval, and triangular neurons. Results were compared to controls and statistically analyzed. The average volume of cell nuclei was larger in group I (549.9 μm) and group II (513.0 μm) than in controls (373.0 μm). The difference was most pronounced and statistically significant in nuclei of multipolar neurons and less so in the other types of neurons. The nuclei were oval in shape in 6.6% of group I, 2.8% of group II, and 1.3% of controls; slightly oval in 50.1%, 56.7%, and 46.6% respectively, and round shaped in 42.3%, 40.5%, and 52.1% respectively. The difference in shape was most pronounced in multipolar neurons of group I and II. The location of nucleoli was more often acentric in group I (65.4%) than in controls (50.8%). In group II central location of the nucleolus predominated in all types of neurons. It is concluded that the morphological changes in cells of the subthalamic nucleus (increase in volume and oval shape of nuclei, acentric location of nucleoli) in group I indicate an increase in cellular activity in animals responding with convulsions to administration of this pesticide.

75-1392. Vela-Muzquiz, R.; Kasper, P. (Dep. Biol. Sci., North Texas State Univ., Denton, TX), **Effecto del pentachlorofenol sobre la flora microbiana de selos seleccionados. [Effect of pentachlorophenol on the microbial flora of selected soils.]** *Microbiol. Espan. 26: 21-30; 1973.* (14 references) (Spanish)

Microbial flora of fertile soils is incapable of utilizing pentachlorophenol (PCP) or its sodium derivative (Na-PCP) as a sole source of carbon. When these substances (0, 20, 40, and 80 mg/l.) were added to nutrient broth or soil cultures with yeast extract, the growth of soil bacteria, fungi, actinomycetes, etc. was completely inhibited. Flavobacterium and Enterobacter were found to be capable of growing in the presence of PCP and Na-PCP but did not degrade the compounds. It appears that the soil microflora do not play an important role in the degradation of PCP or Na-PCP.

75-1393. Ulasevich, E. I.; Kharachenko, S. M.; Yakubovsky, S. V.; Manko, Yu. P. (Ukr. Agric. Acad., Kiev, USSR). **Vpliv linuronu na mikrofloru chornozemu i rizosferi kukurudzi ta soy. [Effect of linuron on microflora of chernozem and rhizosphere of maize and soybean.]** *Mikrobiol. Zh. (Kiev) 35(2): 231-235; 1973.* (8 references) (Ukrainian)

Results are presented on the effect of herbicide linuron on the quantitative composition of mold fungi, actinomycetes, and bacteria as well as the individual groups in soil, and in maize and soybean rhizosphere in field experiments with deep low-humic chernozem. Doses of 3.3 and 4.1 kg per hectare of linuron were applied to increase the bacterium content in soil and plant rhizosphere as well as the fungi quantity in soil and soybean rhizosphere. Development of fungi near the maize roots was inhibited by the effect of linuron. The herbicide decreased the number of actinomycetes in soil and plant rhizosphere and produced no particular effect on the development of azotobacter and oligonitrophilous bacteria.

75-1394. Alexandri, A. V.; Nagler, M.; Alexandrescu, S. (Natl. Inst. Plant Prot., Bucharest, Romania). **Actiunea unor insecticide cloroderivate asupra florei bacteriene din sol. [Effects of some chlorinated insecticides on the bacterial flora of the soil.]** *Microbiologia (Bucuresti) 1: 463-466; 1970.* (3 references) (Romanian)

Soil treatment with Aldrin 20 (4 and 8 μg/ha), Heclotox 3 (2.1 and 4.2 g BHC/ha), Detox 5 (3.5 and 7 *p,p'*-DDT/ha) was carried out in potato and pea crops. Observations were made 24 hours, 20 and 45 days after the treatment, taking into account only the viable bacteria (which were not identified as to their taxonomic position). Results showed that the total number of bacteria in the soil was not significantly influenced by the insecticides, even when the rates far exceeded the normal ones.

75-1395. Ehrenberg, L.; Hiesche, K. D.; Osterman-Golkar, S.; Wennberg, I. (Wallenberg Lab., Univ. Stockholm, Stockholm, Sweden). **Evaluation of genetic risks of alkylating agents: tissue doses in the mouse from air contaminated with ethylene oxide.** *Mutat. Res. 24(2): 83-103; 1974.* (88 references)

The fate of ethylene oxide in mice exposed to air contaminated with the compound in concentrations which occur in the human environment was studied. The possibility of using tissue doses as a basis of quantitative estimates of genetic risk of electrophilic compounds in the environment was explored. The degree of alkylation

of proteins was used to determine tissue doses D*t*, that is, the concentration of free alkylating agent, integrated over time, in resting male mice exposed for 1-2 hr to air containing 1-35 ppm ethylene oxide. Exposure doses were 0.03-2% of LD50. Results indicate absorption of all ethylene oxide in alveolar ventilation, a rapid distribution to all organs, and a rapid detoxication and excretion. D*t* was proportional to the exposure dose within the range studied. In most organs, including the testes, the D*t* is about 0.5 μM-hr/ppm-hr exposure. The degree of alkylation of DNA agreed with those expected from the doses determined. Expressing genetic risks of environmental chemicals in the frame of reference of radiation hazard facilitates comparison and summation of risks of various origins. On the basis of dose-effect curves of ethylene oxide and X-rays in barley, a tissue dose of ethylene oxide in man of 1 μM-hr may be provisionally set equal to 80 rad of low LET radiation. Allowing for the difference in alveolar ventilation between mouse and man, this would mean that epoxide operators working at 5 ppm ethylene oxide for 40 hr/week receive a weekly gonad dose of ethylene oxide amounting to about 4 "rad-equivalents". This risk estimate is shown to be realistic.

75-1396. Beattie, K. L.; Kimball, R. F. (Oak Ridge Grad. Sch. Biomed. Sci., Univ. Tennessee, Oak Ridge, TN 37830). **Involvement of DNA replication and repair in mutagenesis of *Haemophilus influenzae* induced by *N*-nitrosocarbaryl.** *Mutat. Res. 24(2): 105-115; 1974.* (10 references)

A temperature sensitive DNA synthesis mutant of *Haemophilus influenzae* (strain *dna*9) was treated with the *N*-nitroso compound, *N*-nitrosocarbaryl, then incubated at acceptable and excessive temperatures, 36 and 41 degrees, respectively. Lysates were made at various times and used to transform a second culture to novobiocin resistance, a measure of the extent of mutation fixation. At the acceptable temperature, mutation fixation continued almost linearly during at least half of the first round of DNA replication after *N*-nitrosocarbaryl treatment. In the absence of DNA replication (41°C), most, but not all, of the mutation fixation was eliminated. The nonreplicative type of mutation fixation was greater after treatment with a higher *N*-nitrosocarbaryl concentration. The data indicates that premutational lesions occur over the entire chromosome and that the bulk of the mutation fixation requires DNA replication, but that a process independent of replication, quite possibly an error prone repair system, also is responsible for part of the mutation fixation in cells exposed to alkylating agents. When strain *dna*9 was treated with *N*-nitrosocarbaryl and then incubated at 41° for some time before being grown at 36°, a large decrease in the final mutation frequency was seen. This suggests that a repair mechanism still functional in the absence of DNA replication is capable of removing premutational lesions from *H. influenzae* DNA.

75-1397. Georgian, L. (Victor Babes Inst. Pathol. Med. Genet., Bucharest, Romania). **The comparative cytogenetic effects of aldrin and phosphamidon.** *Mutat. Res. 31(2): 103-108; 1975.* (21 references)

An organochlorinated pesticide (aldrin) and an organophosphorus one (phosphamidon) were administered in human lymphocyte cultures, and the cytogenetic effects were related to the compound concentration. The comparative estimation of the number and type of chromosome aberrations observed in the treatments with various doses of drugs permits a number of statements. The aldrin showed a narrow range of clastogenic doses, between 19.125 and 38.25 μg/ml. Since these doses are near the limit for cell survival, the observed chromosome lesions are probably not perpetuated in other abnormal cells. Comparatively, the range of phosphamidon clastogenic doses is very large, scattered between 1.9 and 122 μg/m. Since, in the phosphamidon treatments, the cellular death begins at a concentration above 244 μg/ml, the chromosome aberrations, induced especially by the low doses, could be maintained in other abnormal cells. In a smaller number of experiments, chromosome examinations were performed after intraperitoneal injections of the drugs into rats and mice, 24 hr before harvesting of the bone marrow. The administered doses were low, as compared with those of the experiments in vitro: the minimal doses inducing chromosome aberrations in vivo were, in the aldrin treatment 9.56, and in the phosphamidon treatments, 0.07 μg/g body weight. In the experiments both in vitro and in vivo, the analysis of the frequencies of the abnormal cells and of the chromosome lesion types support the existence of the dose-response correlation. The genetic peril due to low doses of pesticides with a general weak toxic effect is discussed. (Author abstract by permission)

75-1398. Murata, M.; Tomita, K. (Agric. Dep., Sankyo Co. Ltd., Tokyo, Japan). **[Development and commercialization of a new soil fungicide "Tachigaren."]** *Nikkakyo Geppo (Mon. Rep. Jpn. Chem. Ind. Assoc.) 1975 (3): 146-152; 1975.* (Japanese)

Acute p.o. toxicity of a new fungicide, Tachigaren, 3-hydroxy-5-methylisoxazole (hymexazole), mainly used for soil treatment to protect rice seedlings against damping off, expressed as LD50, was 2148, 1968, 4678 and 3909 mg/kg respectively to male and female mouse and male and female rat. TLm (48 hr) to young carp is 165 ppm. Translocation of the a.i. from root to leaf periphery takes about 24 hr. Tachigaren absorbed by plants was easily metabolized into two glucosides, of which the one with the OH group at 3-position bound to glucose retains its fungicidal activity and the other in which the ring nitrogen is bound to glucose shows no such activity. About 97% of tachigaren administered p.o. to rats is excreted in urine as tachigaren and its metabolites; about 93% is excreted within 6 hours. The half-life in living organisms is thought to be about 25 min,

although the peak appears in the kidney at 30 min after administration. After 4 hours the residue in organs became scarcely detectable, hence accumulation by mammals seems unlikely. It was found that 65% was excreted in the urine as the 3-sulfate, 30% as the 3-glucuronide, and 5% as the unchanged form within 24 hours.

75-1399. Takeda, S., Tsukahara, I.; Takaori, S. (Kansai Med. College, Osaka, Japan). **[Action of organophosphorus pesticide on central optical paths.]** *Nippon Ganka Gakkai Zasshi (J. Jpn. Ophthalmol. Soc.) 79(Suppl.): 40; 1975.* (Japanese)

The action of diethyl 2-chloro-1(2,4-dichlorophenyl)vinyl phosphate (chlorfenvinphos) on central optical paths of cats was examined by light-stimulated EPC, induced potentials of optical tract (OT), lateral genu (LG), and visual area of cerebral cortex (VC). The dose rates of chlorfenvinphos were respectively 1, 4, 16, and 64 mg/kg dissolved in oil, and after 0.5, 1, 2, and 3 hr of intraperitoneal administration to the cat, its induced potentials were determined. After chlorfenvinphos administration EPG showed increased width of vibration, and potentials of OT and LG induced by light stimulation showed decreased width of vibration. The induced potential of VC showed increased or decreased amplitudes. Induced potential of LG and VC when OT was electrically stimulated showed increased amplitude after administration of chlorfenvinphos. The action of chlorfenvinphos on induced potentials in the central optical tract was discussed in terms of results and of cholinesterase activity in erythrocyte and brain.

75-1400. Miyata, T.; Saito, T.; Iyatomi, K. (Lab. Appl. Entomol. Nematol., Fac. Agr., Nagoya University, Chikusa, Nagoya, Japan). **[Effect of NS 2662 on the content of adenosine polyphosphate in the muscle of the mouse.]** *Nippon Oyo Dobutsu Konchu Gakkai-Shi (Jpn. J. Appl. Entomol. Zool.) 17(2): 97-100; 1973.* (12 references) (Japanese)

When *O,O*-dimethyl 2,2-dichloro 1-hydroxyethyl phosphonate (NS 2662) was orally administered to mice, the poisoned mice were paralyzed and died without showing characteristic symptoms of organophosphorus poisoning such as severe hyperactivity and convulsions. However, when NS 2662 was injected into the brains through the occipital foramen, the mice showed hyperactivity and convulsions, and died. After ^{14}C-NS 2662, ^{14}C-DMVP (*O,O*-dimethyl 2-monochlorovinyl phosphate), ^{14}C-trichlorfon, or ^{14}C-dichlorvos was administered orally to mice or injected into the brains greater amounts of insecticides were found in the muscles of those orally treated. Maximum muscle insecticide levels, seen after oral NS 2662, were 10^{-4} *M* and 10^{-5} *M* at 30 min and 4 hr after the application, respectively. Adenosine 5′-triphosphate (ATP) content was minimum in the muscle of mice to which NS 2662 had been applied orally. Characteristic symptoms in these poisoned mice without severe hyperactivity and convulsions may be attributed to the lack of ATP which serves as an energy source of muscle movement.

75-1401. Yoshioka, Y. (Dr. Hiraki Sch. of Int. Med., Fac. Med., Okayama Univ., Okayama, Japan). **[Studies on the metabolism of organochlorine pesticides in living organisms Part 1. A study on the distribution of organochlorine pesticides in living organisms by liquid scintillation counter.]** *Okayama Igakkai Zasshi (J. Med. Assoc. Okayama) 86(11-12): 573-579; 1974.* (Japanese)

As a first step in elucidating the mechanism of action of organochlorine pesticides in living organisms, the chronological course of distribution of ^{14}C labeled DDT and ^{14}C labeled dieldrin administered p.o. to mice was determined using a liquid scintillation counter. The dose rates of both pesticides were the same, 2 μCi per mouse. In the case of DDT, the radioactivities in liver, kidney, heart, brain, lung, spleen, and omental adipose tissue showed the same tendency, reaching a maximum of 15848 dpm/g after 48 hours. Then the activity decreased with time (4614 dpm/g after 5 days.) Characteristic persistence in omental adipose tissue was noted. In the group to which unlabeled DDT was first administered daily for 6 days before ^{14}C labeled DDT, the uptake of radioactivity was less than that in the case of administration of only ^{14}C labeled DDT, probably because of the saturation of DDT in the organs and tissues. In the case of ^{14}C labeled dieldrin, the radioactivity showed nearly the same tendency as DDT, except that kidney activity reached 5940 dpm/g after 48 hr and the persistence in omental adipose tissue was less than in the kidney.

75-1402. Yoshioka, Y. (Dr. Hiraki Sch. Int. Med., Fac. Med., Okayama Univ., Okayama, Japan). **[Studies on the metabolism of organochlorine pesticides in living organisms by whole body autoradiography.]** *Okayama Igakkai Zasshi (J. Med. Assoc. Okayama) 86(11-12): 581-586; 1974.* (33 references) (Japanese)

The time change in distribution of orally administered radioactive DDT and dieldrin in living organisms was examined using ICR mice and by the method of whole body autoradiography. The dose rate was 5 μCi per mouse and the film exposure period 30 days. In the case of DDT, the intake of radioactivity into liver, adipose tissue and bone marrow was noticeable after 12 hr. However, after 3 days the accumulation was only noticeable in adipose tissue and after 5 days the amount of radioactivity decreased even in adipose tissue, although a small amount of activity still remained in the other organs. In the case of dieldrin, the intake of radioactivity was noticeable in liver, adipose tissue and bone marrow as well with less intake into brain after 12 hr. After 3 days the trend of persistence was the same as that for DDT and after 5 days the residual activity was

more than that of DDT, especially in adipose tissue, with small residual amounts in other organs. The accumulation of DDT and dieldrin in bone marrow after 12 hr as determined by autoradiography would be an important finding.

75-1403. Tarmas, J.; Stefanska-Sulik, E.; Pieczko-Kuduk, I. (Dep. Zakl. Anat. Inst. Biol. Morphol., Bialystok, Poland). **Morfologiczne zmiany w osrodkowym ukladzie nerwowym swinki morskiej pod wplywem pestycydu Alvit 55. [Morphologic changes in the central nervous system in guinea pigs under the influence of the pesticide Alvit 55.]** *Patol. Pol. 24(2): 295-302; 1973.* (5 references) (Polish)

Mature guinea pigs were fed a single 25 mg/kg dose of the pesticide Alvit 55 (dieldrin) by mouth. Morphologic lesions in the central nervous system after 4, 12, 24, 96 and 192 hours resulted. Changes in the neurons of autonomic nuclei appeared after 4–12 hours. Later, changes were observed in the nuclei of the vestibular system and in the reticular system of the brain stem which after 8 days tended to regress. Irreversible changes persisted in only a few neurons of the dorsal motor nuclei of the vagus nerves and in the macrocellular nuclei of the reticular formation.

75-1404. Pilichowska, J. (Dep. Chem. Ogolnej, Inst. Fizjol. Chem., Szczecin, Poland). **Oddzialywanie pestycydow: nogos G50, MCPA-500 i medzian-50 na oksydaze cytochromowa (E.C.1.9.3.1). [The effect of pesticides Nogos G50, MCPA-500 and Pol-Kupritox-50 on cytochrome oxidase (E.C.1.9.3.1).]** *Pol. Tyg. Lek. 28(44): 1716-1718; 1973.* (6 references) (Polish)

A study is presented on the effects of pesticides with trade names of Nogos G50 (dichlorvos), MCPA-500, and Pol-Kupritox-50 (copper oxychloride) on the activity of cytochrome oxidase derived from bovine myocardium. It was demonstrated by manometric measurements carried out at pH = 7.4 and 25°C that the insecticide Nogos G50 and the herbicide MCPA-500 reduce the activity of cytochrome oxidase as evidenced by decreased ability of oxygen uptake by the studied system. It was found that the inhibitory action of both these substances rises with their rising concentration in the solution. The effect of Pol-Kupritox depends on the level of this fungicide in the solution tested. At concentrations not exceeding 10^{-8} gram-ions of Cu^{2+} the amount of oxygen taken up by the system falls, while with higher concentrations the oxygen uptake rises. It was shown by spectrophotometric measurements that the Cu^{2+} ion derived from Pol-Kupritox oxidase is responsible for the deactiviting action of the fungicides. Increased oxygen uptake by the solution in the presence of higher doses of Pol-Kupritox is due to non-enzymatic processes.

75-1405. Chmielnicka, J.; Markiewicz, K.; Smolinska, E. (Katedry Nauki Srodkack Spozywczych, Lodz, Poland). **Wplyw pestycdow–weglowodorow chlorowanych i zwiazkow fosforooganicznych–na aktywnosc pepsyny soku zoladkowego czlowieka. [The effect of pesticides on the activity of pepsin in human gastric juice.]** *Pol. Tyg. Lek. 28(47): 1844-1847; 1973.* (11 references) (Polish)

Gastric juice samples of varying pH were obtained from inpatients and experiments were carried out with preparations of the pure pesticides DDT, DMDT (methoxychlor), heptachlor, parathion, dipterex (trichlorfon) methyl demeton, and demeton-S-methyl, and with technical preparations of Alvit 55 (dieldrin), Tritox suspension 50 (DDT, lindane, and methoxychlor), liquid Methox 30 (methoxychlor), and Birlane 10 granulated (chlorfenvinphos). DDT was found to inhibit the action of pepsin in vitro in a concentration of 10^{-12} M/ml. Inactivation of this enzyme in the presence of 10^{-8} M/ml of DDT was 68%. DMDT and parathion, as well as Tritox, Methox, Alvit, and Birlane are also strong inhibitors with proteolytic activity from 10^{-6} 10^{-7} M/ml. Heptachlor and dipterex had no effect on enzymatic activity. On the other hand, the demeton compounds showed activation of pepsin in the range of 150-250% of the initial value. It was shown that DDT has a competitive inhibitory action on pepsin. E_1 for DDT was 0.95×10^{-5} M. Parathion is a non-competitive inhibitor of pepsin with a *Ki* of 4.7×10^{-4} M.

75-1406. Krampl, V.; Hladka, A. (Inst. Ind. Hyg. Occup. Dis., Bratislava, Czechoslovakia). **Vplyv sucasneho podania lindanu a fenobarbitalu na stimulaciu pecenovych mikrozomalnych enzymov. [The effect of simultaneous administration of lindane and phenobarbital upon stimulation of microsomal liver enzymes.]** *Prac. Lek. 26(5): 169-172; 1974.* (17 references) (Slovak)

The effect of separately and simultaneously administered lindane and phenobarbital on the stimulation of microsomal liver enzymes was studied in male albino rats. The animals were administered single 80 mg/kg doses of lindane (p.o.) and phenobarbital (i.p.), or 5 daily 20 mg/kg doses of the two substances in the same manner separately or simultaneously. The activity of the microsomal enzyme systems which catalyze the detoxication of EPN and the *O*-demethylation of *p*-nitroanisole was determined quantitatively directly in liver homogenates. Following single and repeated administration, both inducers stimulated the enzymatic activity significantly, regardless of administration jointly or separately. The increase, however, was smaller following simultaneous administration of the two substances than could be expected for the respective substances when administered alone. The findings suggest a competitive attenuation of the stimulation of microsomal liver enzymes by very high doses of lindane and phenobarbital.

75-1407. Bergel, F. (Magnolia Cottage, Bel Royal, Jersey, Channel Islands, England). **Carcinogenic hazards**

in natural and man-made environments. *Proc. R. Soc. London Ser. B 185: 165-181; 1974.* (71 references)

The complex interaction between man and his environment has been explored to some extent; some of the chemical hazards present have resulted from man-made products, for example, insecticides, fungicides, and herbicides. These substances have a wide range of chemical structure. It is suggested that the carcinogenic, mutagenic, or teratogenic effects of these compounds may be functionally connected with the molecular and/or electronic structural features of the compounds themselves and of their cellular targets. It has been discovered that potentially noxious materials undergo certain reactions and transformations as a precondition for their becoming ultimate or proximate agents. The ultimate reaction seems to be displacement attacks of electrophiles on nucleophilic cellular components. Attempts to examine structural relationships are made in the belief that recognizing these similarities between compounds may be helpful in predicting undesirable properties of a compound before these properties manifest themselves in tests or in actual use situations.

75-1408. Ghezzo, F.; Corradini, L.; Guglielmini, C.; Ninfo, V. (Inst. Hyg., Univ. Ferrara, Ferrara, Italy). **L'azione tossica dei ditiocarbamati su sistemi enzimatici. [Toxic effects of dithiocarbamates on enzymatic systems.]** *Quad. Sclavo Diagn. Clin. Lab. 8(1): 485-494; 1972.* (17 references) (Italian)

The toxicity of dithiocarbamates on mammalian systems, and especially on enzymatic systems is examined. The results of two studies on the long-term effects of zinc dimethyldithiocarbamate (Ziram) on rabbit and mouse plasma alkaline phosphatase levels are reported. Levels decreased in the rabbits and increased in the mice, significantly compared to the control groups. Comments are made on the effect of several types of dithiocarbamates on enzymatic systems, and especially blood and tissue alkaline phosphatases.

75-1409. Sato, N.; Yakawa, K.; Nishio, Y.; Uemura, K. (First School of Pathol., Iwate Med. Coll., Morioka, Iwate, Japan). **[The alteration of mouse liver by long-term administration of 3-amino-1,2,4-triazole and propylthiouracil.]** *Nippon Gangakkai Kiji (Proc. Jpn. Cancer Assoc.) 33: 49; 1974.* (Japanese)

One group of female DDN mice were given an aqueous solution of 2000 ppm of 3-amino-1,2,4-triazole (amitrole) ad lib. and fed daily with diet containing 0.2% propylthiouracil. Another group (II) of 33 female DDN mice were given the same solution ad lib. and fed with standard daily diet. After 400 days of feeding the livers of the surviving 5 (I) and 7 (II) mice were examined microscopically and electron-microscopically. All the livers of group I mice swelled conspicuously, were generally hard with irregular surface, and showed innumerable greyish-white nodules on surface and cross-section and absence of normal liver tissue. Histologically, hepatocellular funiculi in the nodules were random with polymorphous liver cells having irregular nuclei. The livers of group II showed no enlargement and had a smooth surface. Even on a cross-section, point-like small nodules were noticed only occasionally. The comparison of groups I and II showed clearly the proliferation of tumorous liver cells due to 3-amino-1,2,4-triazole.

75-1410. Tsuda, H.; Hirose, M.; Tatematsu, M.; Fukushima, A.; Takahashi, M. (First Sch. Pathol., Fac. Med., Nagoya Munic. Univ., Nagoya, Aichi, Japan). **[The effect of 3-amino-1,2,4-triazole on the transplanted thyroid gland and subtotally excised thyroid gland of rats.]** *Nippon Gangakkai Kiji (Proc. Jpn. Cancer Assoc.) 33: 49; 1974.* (Japanese)

Three groups of Wistar rats were respectively treated in various ways. (I) Isograft group: thyroid tissue was removed with trachea just after birth and transplanted subcutaneously into sibling rats. (II) Autograft group: both thyroid glands of a female rat were removed and transplanted subcutaneously. (III) Subtotal thyroidectomy group: both thyroid glands of a female rat were removed as completely as possible to the naked eye. At 5 wk after treatment, the rats were given an aqueous solution containing 2500 ppm of 3-amino-1,2,4-triazole (amitrole) from the waterer. Although papillate proliferation of follicular epithelium and disrupted capsules were found, no metastatic focus was found in group I and II. Five out of 30 rats of group I showed hepatic cholangiofibrosis. Although there was regeneration of thyroid tissue with marked fibrosis of the stroma in group III , no disrupted veins, no adenomatously proliferated focus nor formation of metastatic focus was found. In short, 3-amino-1,2,4-triazole exerted essentially the same effect on rats with normal thyroid glands as on those with transplanted thyroids and on those with thyroid glands subtotally removed.

75-1411. Kuwabara, N.; Takayama, S. (Sch. Pathol., Fac. Med., Juntendo Univ., Tokyo, Japan). **[Comparison of histogenesis of liver in mice administered respectively BHC, DDT, and 2,7-FAA.]** *Nippon Gangakkai Kiji (Proc. Jpn. Cancer Assoc.) 33: 50; 1974.* (Japanese)

Groups of ICR male mice were respectively administered BHC (600 ppm in daily food), DDT (250 ppm in daily food), and 2,7-FAA (250 ppm in daily food). Other groups were fed with 2,7-FAA in food for a period and then fed BHC or DDT in food. Their pathological states were morphologically examined as well as their production of alpha-fetoprotein. In the BHC group focal hepatic cellular hyperplasia appeared (non-neoplastic or non-hyperplastic) and some foci changed to hyperplastic nodules and hepatic cellular adenoma. In the DDT group, a non-neoplastic and hyperplastic pathologic state appeared first and then adenoma was formed. In the 2,7-FAA group, non-neoplastic or hyper-

plastic nodules appeared at first and then changed to adenoma and hepatocellular carcinoma with sarcoma in a few instances. In the group first administered 2,7-FAA and then given BHC or DDT, nearly the same phenomena were observed in the group fed 2,7-FAA alone. The production of alpha-fetoprotein was positive only in the case of hepatoma in the 2,7-FAA group and negative in the BHC group and the DDT group.

75-1412. Nagasaki, H.; Aoi, H.; Makiura, S.; Aoki, Z.; Arai, M.; Konishi, Y.; Ito, N. (Sch. Hyg., Nara Med. College, Nara, Japan). **[Characteristic features of hepatoma of mice due to α-BHC and several factors of carcinogenesis.]** *Nippon Gangakkai Kiji (Proc. Jpn. Cancer Assoc.) 33: 78; 1974.* (Japanese)

The effects of animal strain, sex, and other factors on liver tumorigenesis in the mouse, and the reversibility and non-reversibility of tumor induction were examined in three experimental series. In the first series 40 male and 40 female mice of the ddy, ICR, DBA2, C57BL6, and C3H-HE strains were fed a diet containing 500 ppm of α-BHC for 24 weeks. Second, 160 male mice of the ddy strain were fed 500 ppm of β-BHC, 1000 ppm of *p*-hydroxypropiophenone, 67 ppm of 3-methylcholanthrene, or 600 ppm of α-naphthylisothiocyanate singly or in combination for 24 weeks. In the third series 650 ddy mice of both sexes were fed diet containing 500 ppm of α-BHC for 24 and 36 weeks, respectively, then fed standard diet for 12, 24, and 36 weeks. The histopathological morbidities in the three groups were compared. The morbidity rate of hepatic tumors decreased in the order of ddy, ICR, C3H, DBA and C57BH-strain. Males showed higher morbidity than females. The three compounds showed no effect on liver tumorigenesis due to α-BHC. The increase in liver weight gain slowed after the suspension of BHC administration; however, it showed a renewed increase after 12 weeks due to growth of hepatic tumor. It was concluded that the carcinogenicity of α-BHC in mouse liver was established and that in some instances the nodular hypertrophy changed irreversibly to hepatoma.

75-1413. Ranke-Rybicka, B.; Stanislawska, J. (Dep. Communal Hyg., Natl. Inst. Hyg., Warsaw, Poland). **Zmiany w zespolach perifitonowych wywolane dzialaniem pestycydow fosforoorganicznych (malation, foschlor). [Changes in periphyton complexes following application of organophosphorus pesticides (malathion, foschlor).]** *Rocz. Ranstw. Zakl. Hig. 23(2): 137-146; 1972.* (17 references) (Polish)

The effect of the organophosphorus pesticides Sadophos and Foschlor, and of their chemically pure crystalline active ingredients 95% malathion and 97.85% trichlorfon respectively on periphyton communities colonizing glass slides in aquaria was investigated under natural atmospheric conditions. Following the use of the pesticides (from 2 to 12.54 mg/l of chemically pure substances), a diminution of the number of organisms was noted three days after application at a concentration of 7.42 ml/liter of 30% Sadophos and 5.71 mg per liter of 20% Foschlor. Restitution of the aqueous organisms took two weeks. Protozoa were the most sensitive organisms while algae were the least affected. In the period of regeneration of periphyton complexes many polysaprobic organisms appeared, e.g., *Glaucoma pyriformis* Ehr, and *Vorticella microstoma* Ehr, replacing the mesosaprobes *G. scintillaus* Ehr, and *V. convalaria* L.

75-1414. Nikonorow, M.; Ludwicki, J. K. (Zakl. Badania Zywnosci i Przedmiotwo Uzytku, Natl. Inst. Hyg., Warsaw, Poland). **Wplyw butylo-hydroksytoluenu (BTH) i kaptanu na aktywnosc kwasnej fosfatazy w zaleznosci od sposobu zywienia zwierzat. [The influence of BHT, captan, and low-protein diets on the activity of acid phosphatase in rats.]** *Rocz. Panstw. Zakl. Hig. 23(3): 253-257; 1972.* (11 references) (Polish)

In the serum and liver of male and female Wistar strain albino rats, enzyme activity was determined. The effects of various levels of protein in the diet and additions of captan (0.01% and 0.1%) and BHT (0.5%) on the activity of acid phosphatase were examined. A decrease in the activity of acid phosphatase in the serum of rats of both sexes fed on a protein-deficient diet was observed. There was no effect of captan and BHT on acid phosphatase activity in any of the low protein diets.

75-1415. Wojcik, J. (Dep. Communal Hyg., Natl. Inst. Hyg., Warsaw, Poland). **Badania wplywu foschloru w wodzie do pica na niektore odczyny histochemiczne watroby bialych szczurow w warunkack dlugotwalego doswiadczenia. [Influence of foschlor upon some histochemical liver reactions in white rats under conditions of prolonged experiment.]** *Rocz. Panstw. Zakl. Hig. 23(3): 365-370; 1972.* (15 references) (Polish)

The influence of foschlor (trichlorfon) on the liver tissue of white rats under conditions of chronic intoxication was studied. Five week old female rats were deprived of water and given foschlor solutions of 0.001, 0.01 and 0.1 LD50. The experimental groups, and the control consisted of 10 animals each and were decapitated at the end of experiment. Minor histopathologic lesions were only found in liver tissue following chronic administration of foschlor at the highest concentration (0.1 LD50). The morphologic changes in liver cells consisted of the destruction of the nucleus and cytoplasm. Cells were enlarged, cytoplasm foamy with bright spots, and the nucleus with chromatin and nucleolus were clearly visible. The above morphologic changes were accompanied by a minor RNA and glycogen deficiency, decreased activity of mitochondrial enzymes, minor fatty degeneration and a conspicuous increase in the reactions of acid phosphatase and non-specific esterases. Rat livers in the remaining treatment groups

did not differ morphologically or histochemically from the control animals.

75-1416. Ranke-Rybicka, B. (Dep. Communal Hyg., Natl. Inst. Hyg., Warsaw, Poland). **Badania nad przezywalnoscia kijanek *Rana temporaria* L. Poddawanych okresowemu dzialaniu pestycydow fosforoorganicznych (foschlor, malation). [An inquiry into the viability of tadpoles of *Rana temporaria* intermittently exposed to phospho-organic pesticides (foschlor, malathion).]** *Rocz. Panstw. Zakl. Hig. 23(3): 371-377; 1972.* (19 references) (Polish)

The effects of daily and weekly intoxication by foschlor (trichlorfon) and malathion upon growth and behavior of tadpoles of *Rana temporaria* aged 7 and 30 days were investigated. Foschlor, containing 97.85% trichlorfon (18 mg/l and 1.8 mg/l) and malathion containing 95% of active substance (1.25 mg/l and 0.125 mg/l) were used. Concentration of pesticides in water was assessed on the basis of the 96 hr TLm for small fish with the margin of safety amounting to 0.1 and 0.01. In addition, the influence of a mixture containing 9 mg of foschlor and 0.625 mg of malathion/l, as well as of another containing 0.9 mg foschlor and 0.0625 mg of malathion was also determined. A clear-cut difference in mortality, mobility, and growth between the tadpoles treated for 24 hours and six days, and the control group was found. Few, if any, consequences of intoxication over 24 hours were noted when treatment was discontinued. Intoxication lasting 6 days brought about progressive limitation of mobility of experimental animals which produced deaths up to 100% or irreversible stunting during a 15 day follow-up.

75-1417. Zadrozinska, J. (Zak. Badania Zywnosci i Przedmiotow Uzytku, Natl. Inst. Hyg., Warsaw, Poland). **Badanie procesow przemiany fentionow w wybranych roslinach. [A study of fenthion breakdown in selected plants.]** *Rocz. Panstw. Zakl. Hig. 23(4): 409-416; 1972.* (8 references) (Polish)

Broken bean plants and black currants were treated with Lebaycid (manufactured by Bayer and containing 50 per cent of fenthion). Concentrations of 0.15% lebaycid solution were sprinkled on the broken beans and currants. Another group of bean plants was watered with a 1% solution. Samples for study were taken at two-day intervals until total decay of fenthion metabolite residues. Assessment of insecticides was done using thin-layer chromatography coupled with the enzymatic method. In the plants under treatment, aside from fenthion (the presence of which has been encountered only for a short period following the experimental procedure) four metabolites were found: fenthion sulfoxide and sulfone, and fenthoxon sulphoxide and sulfone. In the watered beans only the fenthion metabolites, but no fenthion, were found. Sulfoxides were the main metabolities. Irrespective of the kind of plant used and the treatment procedure, fenthion sulfoxide was the most durable compound, still being found after 30 days in the sprinkled beans, 42 days in the watered beans, and 64 days in black currants following treatment.

75-1418. Bankowska, J.; Bojanowska, A. (Dep. Toxicol. Sanit., Natl. Inst. Hyg., Warsaw, Poland). **Odtruwanie insektycydu fosforoorganicnego w wyniku indukcji enzymatcznej powodowanej przez herbicydy mocnikowe. [Detoxification of organophosphorus insecticide following enzymatic induction caused by urea herbicides.]** *Rocz. Panstw. Zakl. Hig. 23(4): 487-493; 1972.* (7 references) (Polish)

Six-week old Wistar strain male rats were exposed daily to 1/5 and 1/10 LD50 of diuron, linuron, and chlortoluron for five days. On the sixth day the animals were treated with 10 mg/kg body weight doses of parathion and, one hour following the treatment, AChE activity was estimated in blood and brain tissue. In rats which had been exposed to 1/10 LD50 of herbicides (particularly linuron and diuron) a clear-cut diminution of intoxication by parathion was observed. Rats treated with a daily dose of 1/5 LD50 of herbicides yielded no distinct results. It is thought that this dose applied five times damages the enzymatic systems involved in the detoxification of organophosphorus insecticides. The increasing rate of detoxification was accompanied by a significant increase of O-demethylase activity at both levels of treatment.

75-1419. Rybak, M. (Dep. Communal Hyg., Natl. Inst. Hyg., Warsaw, Poland). **Wplyw foschloru podawanego w wodzie na rozwoj osobniczy szczura bialego i jego potomstwo. [The effect of foschlor administered in water on the ontogenetic development of white rats and their offspring.]** *Rocz. Panstw. Zakl. Hig. 24(4): 465-475; 1973.* (26 references) (Polish)

Chemically pure foschlor (trichlorfon) in amounts of 0.001, 0.01, amd 0.1 LD50 was given to rats via their drinking water over 20 hours. Forty females and 12 males (4 groups of 10 females and 3 males) were poisoned while one group served as control. In the fourth month of poisoning the level of blood cholinesterase activity was measured in the females and vaginal smears were taken twice daily. After mating and birth of the next generation the animals from the new generation were divided into similar groups as their parents and similar investigations done. The experiment included the third generation. Data noted included: the number of pregnancies, the number of births, neonatal mortality, time of hair growth, and opening of eyes. Body weight was determined in the third and fourth months of life, and then at intervals of 2 weeks. The amount of foschlor solution used in relation to body weight was also noted. Results included a significant decrease of blood cholinesterase activity in all generations in groups obtaining

about 0.01 and 0.1 of LD50. The number of births in the third generation group receiving the highest dose of the poison was significantly lower. Neonatal mortality was high in the group receiving the highest dose of the poison as compared with controls and its rate rose with every successive generation. The body weight of rats in the same group was lower in all generations starting from the 4th week of life. The remaining indices such as regularity of sexual cycle, number of pregnancies, time of appearance of hair growth and eye opening, and the body weight of mature rats were not significantly different from the same indices in control animals. The 0.001 LD50 (nearly 5.1 mg/l) dose of foschlor did not cause significant changes in the tested indices of the rats. The doses of nearly 0.01 LD50 (about 49.1 mg/l) and 0.1 LD50 (about 513 mg/l) had a distinct harmful effect with a marked fall in cholinesterase activity and a high mortality rate in young rats.

75-1420. Bankowska, J.; Bojanowska, A. (Dep. Toxicol. Sanit., Natl. Inst. Hyg., Warsaw, Poland). **Indukujace dzialanie diuronu i linuronu w krotkookresowym doswiadczeniu chroniczynm na dojrzalych szczurach. [Induction effect of diuron and linuron in short term chronic experiments on adult rats.]** *Rocz. Panstw. Zakl. Hig. 24(4): 493-505; 1973.* (14 references) (Polish)

Over 9 weeks, 6 groups of twelve 4-month-old male rats were kept on the following diets: 500 ppm diuron, 5000 ppm diuron, 500 ppm linuron, 5000 ppm linuron, and 50 ppm DDT. After one, three, six, and nine weeks of intoxication three rats from each group were killed and the activity of *O*,*N*, and *S*-demethylases was determined in postmitochondrial supernatant of the liver. The intake of food and the changes in body weight and the relative weight of the liver were measured during the course of the experiment. Of all demethylating systems tested *O*-demethylase was found to be the most characteristic. In the case of both preparations the activity of this system reached a maximum value in the third week of the experiment, followed by a sudden drop and disappearance of the stimulating effect of diuron and linuron. Less characteristic changes of activity were observed in the remaining enzymatic systems.

75-1421. Beskid, W.; Klos, H.; Suwala, Z.; Szyszkowska, A.; Wojcik, J. (Dep. Pathophysiol., Komorki Med. Cent., Warsaw, Poland). **Wplyw malationu na obraz histochemiczny i niektore odczyny histochemiczne watroby szczura. [The effect of malathion on the morphological structure and certain histochemical reactions in rat liver.]** *Rocz. Panstw. Zakl. Hig. 24(5): 529-537; 1973* (13 references) (Polish)

Young male rats weighing 1000 g received malathion in amounts of 0.001, 0.01, 0.1, or LD50 through a gastric tube (the LD50 for rat being about 1200 mg/kg/24 hr). In animals receiving the pesticide in amounts of 0.01 and 0.1 LD50, hepatocellular damage was observed. Findings included blurring of the lobular structure of the liver, loosening of parenchyma, necrosis of cells, and accumulation of Browicz-Kupffer cells in portal spaces. In fragments of damaged liver taken for investigations, a decrease in RNA and glycogen, and fatty degeneration of the cytoplasm were observed in the damaged cells. The morphological changes were associated with increased reaction for acid phosphatase and marked decrease of the activity of non-specific esterases. Increased intensity of changes was observed with increasing doses of the malathion in comparing histochemical reactions and morphological findings in the animal cells.

75-1422. Bankowska, J.; Bojanowska, A. (Dep. Toxicol. Sanit., Natl. Inst. Hyg., Warsaw, Poland). **Zmiany ultrastruktury komorek watroby szczura pod wyplywem niektorych herbicydow. [Ultrastructural changes of rat liver cells caused by certain herbicides.]** *Rocz. Panstw. Zakl. Hig. 24(6): 371-376; 1973.* (5 references) (Polish)

1/10 LD50 of diuron, linuron and chlortoluron were given through a gastric tube for 5 days to nine rats (three for each compound). A fourth group was made the control. On the sixth day the rats were decapitated and their livers isolated. Determinations of *O*-demethylase activity and histological investigations were made on each liver. Electron microscopic investigations disclosed considerable changes in the cytoplasm. Large spaces filled with amorphous masses appeared. It was thought that they developed as a result of rupture and fusion of vesicles in the smooth endoplasmic reticulum. These morphological changes were associated with an increase in *O*-demethylase activity determined in postmitochondrial supernatant of liver cells. These changes are considered to show that the urea herbicides investigated belong to the group of drug metabolizing enzyme inducers of the phenobarbital type.

75-1423. Palut, D. (Dep. Toxicol. Sanit., Natl. Inst. Hyg., Warsaw, Poland). **Badania mechanizmow detoksykacyjnych insektycydow fosforoorganicznych i karbaminianowch. [Studies on the detoxication mechanisms of organic phosphorus and carbamate insecticides.]** *Rocz. Panstw. Zakl. Hig. 24(6): 649-665; 1973.* (35 references) (Polish)

The metabolic pathway of bromophos-^{32}P was studied using postmitochondrial supernatant of the liver of rat, pig and man in the presence of NADPH and GSH. The metabolism of carbaryl-^{14}C was studied using cell preparations of two animal species and man with NADP, UDPGA, and substrates of PAPS synthesis. The metabolism and excretion of carbaryl-^{14}C were also determined in vivo in rats. For comparison of qualitative and quantitative changes in these insecticides the radioactivity was measured in postmitchondrial supernant of rat. These were obtained and stored under the same

conditions as studied on fragments of porcine and human livers. It was found that the process of degradation of bromophos-^{32}P in the presence of GSH probably occurs at the same rate in the livers of both rat and man. On the other hand, a particularly low degree of activity of transmethylases cooperating with GSH was found in the postmitochondrial supernatant of porcine liver.

75-1424. Beskid, M.; Klos, H.; Suwala, Z.; Szczyszkowska, A.; Wojcik, J. (Dep. Communal Hyg., Natl. Inst. Hyg., Warsaw, Poland). **Wplyw malationu na niektore odczyny enzymow oddechowych i ultrastrukture mitochondriow watroby szczura. [The effect of malathion on certain reactions of respiratory enzymes and ultrastructure of mitochondria in rat liver.]** *Rocz. Panstw. Zakl. Hig. 24(6): 741-748; 1973.* (11 references) (Polish)

Thirty Wistar rats were given malathion intragastrically in doses of 0.001, 0.01 and 0.1 LD50 over nine months. Ten rats comprised a control group. Morphological lesions in liver parenchyma were observed following the administration of malathion in doses of 0.01 and 0.1 LD50. In histological sections of the damaged liver a fall in the activity of respiratory enzymes,succinic dehydrogenase, α-glycerol oxidase, and cytochrome oxidase was found. A slight reduction in the activity of lactic dehydrogenase was also observed. Electron microscopy revealed lesions of mitochondria and endoplasmic reticulum in the livers of rats receiving malathion in doses of 0.1 LD50.

75-1425. Bankowska, J.; Bojanowska, A. (Dep. Toxicol. Sanit., Natl. Inst. Hyg., Warsaw, Poland). **Indukujace dzialanie linuronu i chlortoluronu w krotkookresowym doswiadczeniu chronicznym na mlodych szczurach. [Inductive action of linuron and chlortoluron in short-term chronic experiment on young rats.]** *Rocz. Panstw. Zakl. Hig. 25(1): 9-21; 1974.* (13 references) (Polish)

Five groups of rats (25 in each) received food over 13 weeks containing the following preparations: controls 0, linuron 500 ppm, linuron 5000 ppm, chlortoluron 500 ppm, chlortoluron 5000 ppm. Five rats in each group were killed after the first, third, sixth, ninth, and thirteenth weeks of intoxication and the activity of *O*-, *N*-, and *S*-demethylases was determined in the postmitochondrial supernatant of their livers. The intake of food, changes in the body weight of rats, and relative weight of the liver were followed during the course of the experiment. The effect of the investigated herbicides on the activity of *O*-, *N*-, and *S*-demethylases was a tendency of these enzymatic systems to show periodic rises in activity which may be regarded as a particular form of transient stimulation.

75-1426. Fonberg-Broczek, M. (Dep. Food and Produce, State Dep. of Hyg., Warsaw, Poland). **Badania wplywu wybranych pestycydow na actywnosc cytochromu P-450 i UPD-glukuronylotransferazy "in vitro". [The effect of selected pesticides on the activity of cytochrome P-450 and UDP-glucuronyltransferase in vitro.]** *Rocz. Panstw. Zakl. Hig. 25(6): 655-662; 1974.* (15 references) (Polish)

The in vitro effect of 20 pesticides in concentrations of 500 and 50 or 300 and 30 μg/ml on the activity of UDP-glururonyltransferase and cytochrome P-450 dependent *O*-demethylase of *p*-nitroanisole from rat and mouse liver microsomes was investigated. The metallo-organic pesticides zineb, maneb, ziram, ferbam, phenylmercuric nitrate and acetate, and mercuric acetate had a strong inhibitory effect on both enzymes. Aldrin, dieldrin, heptachlor, endrin, endosulfan, and chlordane had no effect on UDP glucuronyltransferase; chlorpropham, captafol, and topsin (thiophanate) had a weak inhibitory effect. Aldrin, dieldrin, heptachlor, chloropropham, and piperon yl butoxide in 300 μg/ml concentration inhibited the rate of *O*-demethylation of *p*-nitroanisole by 30-50%. DDT had no effect on either enzyme. Ethylmercuric chloride and the other mercurial organic pesticides modified the optical difference spectrum of cytochrome P-450 produced by aniline.

75-1427. Chmielewski, K. (Zakl. Chem. Ogolnej, Inst. Fizjol.-Chem., Pomorskiej Adad. Med., Szczecin, Poland). **Wplyw kwasu alfa-lipoinowego na dzialanie toksycznych zwiazkow w niektorych ukladach enzymatycznych. [Influence of alpha-lipoic acid on the action of toxic compounds in certain enzymatic systems.]** *Rocz. Pomorsk. Akad. Med. Szczeninie Suppl. 4: 125-131; 1972.* (6 references) (Polish)

Experimental results are presented dealing with the influence of α-lipoic acid (LA) on the toxic action of fungicides: Zaprawa Nasienna Uniwersalna (ZU), Zaprawa Nasienna R (ZR), and Ceresan (C) (organic mercury compounds) on the hydrogen peroxide decomposition process in the presence of ox liver catalase and hemolyzed human blood. A gasometric method is used. The most effective antitoxic action of LA is recorded for C; it is found to be less effective for ZR and least for ZU, while the toxic action of the fungicides in question increases in the order ZR < C < ZU. The probable mechanisms of these actions are discussed.

75-1428. Casida, J. E.; Gray, R. A.; Tilles, H. (Div. Entomol. Parasitol., Univ. California, Berkeley, CA 94720). **Thiocarbamate sulfoxides: potent, selective, and biodegradable herbicides.** *Science 184(4136): 573-574; 1974.* (9 references)

Sulfoxidation of thiocarbamates yields a new class of chemicals having increased herbicidal activity along with greater tolerance of corn and soybeans in greenhouse tests. However, their thermal stability is not favorable. These sulfoxides are intermediates in the mammalian metabolism of thiocarbamates, being formed by liver microsomal oxidases and cleaved in a system

consisting of glutathione and a soluble enzyme from liver. (Author abstract by permission)

75-1429. Matsumura, F. (Dep. Entomol., Univ. Wisconsin, Madison, WI). **Microbial degradation of pesticides.** *IN: Survival in toxic environments, M. A. Q. Khan and J. P. Bederka, Jr.,eds., Academic Press, New York, 1974, p. 129-154.* (56 references)

Studies on the microbial degradation of pesticides are summarized including reactions involved in detoxication and in conversion of pesticides to toxic terminal residues. The metabolic activities causing detoxication of pesticidal chemicals include hydrolytic processes, oxidative activities, and reductive reactions including dechlorination reactions. Metabolic conversion of pesticidal chemicals can produce compounds of higher toxicities and stability. These chemicals are called terminal residues. The rearrangement process of epoxy rings to form ketones, aldehydes, and alcohols is a characteristic of the isomerization reaction and common to endrin and dieldrin. The epoxidation process of cyclodiene insecticides such as heptachlor and aldrin produces more stable and more toxic terminal residues. The system is omnipresent among microbial, plant, and animal systems. Other systems include oxidation of thioethers to sulfoxides and sulfones and the *N*-dealkylation of alkylamines. There are a few examples of hydrolytic processes forming terminal residues such as the epoxy-ring opening of cyclodiene insecticides and the hydrolyses of organophosphates and carbamates. Other reactions include selective degradation, formation of DDCN, and the alkylation reaction on heavy metal ions.

75-1430. Knowles, C. O. (Dep. Entomol., Univ. Missouri, Columbia, MO 65201). **Detoxication of acaricides by animals.** *IN: Survival in toxic environments, M. A. Q. Khan and J. P. Bederka, Jr., eds., Academic Press, New York, 1974, p. 155-176.* (56 references)

Studies on the metabolic transformation of acaricide chemicals by animals are reviewed. The specific acaricides include: parathion, formetanate, aldicarb, endosulfan, chlorobenzilate, chloropropylate, acarol, dicofol, tetrasul, ovex, fluenethyl, dinobuton, oxythioquinox, chlordimeform, amd banamite. In some cases metabolic attack on the acaricide molecule results in the formation of toxic products. The majority of the biotransformations results in the formation of innocuous polar compounds. The transformations can be regarded as detoxication mechanisms and are important in the survival of animals exposed to acaricides. The detoxication mechanisms include hydrolysis, aromatic hydroxylation, aliphatic hydroxylation, dealkylation, reduction, acetylation, and glycoside and ethereal syntheses.

75-1431. Khan, M. A. Q.; Stanton, R. H.; Reddy, G. (Dep. Biol. Sci., Univ. Illinois, Chicago Circle, Chicago, IL.). **Detoxication of foreign chemicals by invertebrates.** *IN: Survival in toxic environments, M. A. Q. Khan and J. P. Bederka, Jr., eds., Academic Press, New York, 1974, p. 177-201.* (91 references)

The principal detoxication and conjugation systems of insects and other invertebrates are reviewed. The detoxification systems can be located in the cytoplasmic fluid (soluble enzymes) or may be bound to the endoplasmic reticulum (microsomal enzymes). The soluble enzymes include: DDT-dehydrochlorinase, esterases, and reductases. The microsomal enzymes may include some of the esterases and conjugation systems, but they mainly consist of the multi-function oxidases. Both enzyme systems are capable of catalyzing various types of conjugations of a xenobiotic with endogenous molecules. After a foreign chemical is initially metabolized by one or more detoxication systems, the metabolite can be conjugated with a carbohydrate, phosphate, peptide, etc. before it is excreted. The conjugation reactions are more rapid than the detoxication reactions.

75-1432. Adamson, R. H.; Sieber, S. M. (Lab. Chem. Pharmacol., Natl. Cancer Inst., Natl. Inst. of Health, Bethesda, MD 20014). **The disposition of xenobiotics by fishes.** *IN: Survival in toxic environments, M. A. Q. Khan and J. P. Bederka, Jr., eds., Academic Press, New York, 1974, p. 203-211.* (16 references)

The mechanisms for termination of xenobiotic activity in fishes are discussed. They include biliary excretion, renal excretion, gill excretion, and metabolism. Xenobiotic metabolism occurs by both microsomal and nonmicrosomal enzymes. Reduction of the 4-nitro group of parathion to a metabolite with anticholinesterase activity took place by a liver enzyme system in sunfish and bullheads. Hydrolysis is one pathway utilized by fish in metabolizing xenobiotics. It is confined to esters and amides and is an important reaction in the inactivation and quite often the activation of drugs. It is of particular importance in the metabolism and detoxication of insecticides, especially the organophosphate insecticides. The hydrolysis of some of the insecticides by various fish is known. Various xenobiotics are excreted across the dogfish gill to a limited degree. In both DDT and sulfadiazine, 1% of the dose is excreted per hour.

75-1433. Hodgson, E. (Dep. Entomology, North Carolina State Univ., Raleigh, NC). **Comparative studies of cytochrome P-450 and its interaction with pesticides.** *IN: Survival in toxic environments, M. A. Q. Khan and J. P. Bederka, Jr., eds., Academic Press, New York, 1974, p. 213-260.* (122 references)

Cytochrome P-450 as a general system for detoxication is discussed with special emphasis on its interactions with pesticides. Cytochrome P-450 and the electron transport enzymes associated with it form a

mixed function oxidase system which in many organisms and tissues functions in the detoxication of toxic compounds. The system is adapted for increasing the polarity of a wide range of dietary lipophilic toxicants. Data is tabulated from studies on the interactions of pesticides with the cytochrome P-450 in the hepatic microsomes of mouse, rabbit, rat, and sheep. Studies on the induction of cytochrome P-450 by pesticides are reviewed for DDT and DDT analogs, mirex, and methylenedioxyphenyl compounds in mammals. Mirex is the most effective inducer of microsomal mixed function oxidase activity of any pesticide. It is active at the lowest level tested, 1.0 ppm, in diet of mouse. Distinct forms of cytochrome P-450 are found in genetically different strains of the same species; the characteristics of this are under genetic control.

75-1434. Plapp, F. W., Jr. (Dep. Entomol., Texas A and M Univ., College Station, TX). **Genetics of detoxication systems in insects.** *IN: Survival in toxic environments, M. A. Q. Khan and J. P. Bederka, Jr., eds., Academic Press, New York, 1974, p. 315-332.* (82 references)

The genetics of detoxication systems in insects are discussed and the major known types of resistance genes and their relationship to detoxication are reviewed. Resistance first developed to DDT and related chlorinated hydrocarbons such as DDD (TDE) and methoxychlor. Three major genes, two of which involve detoxication, confer resistance to DDT in the house fly: Deh (DDT dehydrochlorinase), DDTmd (microsomal detoxication), and kdr (knockdown resistance). The first discovered major genetic factor for resistance of organophosphates involved a hydrolytic enzyme known as ali-esterase. Carbamates also exhibit ali-esterase activity. Organophosphate detoxication also occurs by a glutathione-dependent ransferase reaction which cleaves *O*-alkyl ester linkages. The microsomal mixed function oxidase system is important in organophosphate and carbamate resistance. Resistance genes associated with increases in insecticide detoxication are usually inherited in a semidominant manner. Their activity can frequently be blocked by synergists and conversely, can be induced by a wide variety of chemical inducers including many of the insecticides themselves.

75-1435. Perry, A. S. (Cent. for Disease Control, U.S. Pub. Health Serv., Atlanta, GA). **Insecticide resistance in insects and its ecological and economic thrust.** *IN: Survival in toxic environments, M. A. Q. Khan and J. P. Bederka, Jr., eds., Academic Press, New York, 1974, p. 399-445.* (235 references)

The resistance of insects to insecticides is reviewed and the ecological and economic impact is discussed. The most important resistance mechanism is the enhanced ability of the resistant insect to detoxify the insecticide at a faster rate and to eliminate it faster than its susceptible counterpart. Insects resistant to DDT have a greater capacity to metabolize DDT via dehydrochlorination which is mediated by the enzyme DDT-dehydrochlorinase. Resistance to BHC is characterized by more extensive degradation of the molecule by the resistant strain involving glutathione-dependent *S*-alkyltransferase enzymes. The cyclodiene compounds are activated rather than detoxified, and resistance is not due to degradation of these toxicants. Organophosphorus and carbamate insecticides are metabolized by microsomal enzymes requiring NADPH and oxygen and to a lesser extent by hydrolytic enzymes. These biotransformations can result in oxidation to more toxic anticholinesterases and in oxidative and hydrolytic degradation to innocuous derivatives.

75-1436. Yarbrough, J. D. (Dep. Zool., Mississippi State Univ., State College, MS). **Insecticide resistance in vertebrates.** *IN: Survival in toxic environments, M.A.Q. Khan and J. P. Bederka, Jr., eds., Academic Press, New York, 1974, p. 373-397.* (22 references)

Insecticide resistance in vertebrates is discussed with emphasis on the role that membranes may play in the resistance phenomenon demonstrated by mosquitofish (*Gambusia affinis*) to organochlorine insecticides. There was no effect on succinic dehydrogenase activity when intact mitochondria isolated from liver and brain of resistant (R) fish were exposed to endrin concentrations of from 0.0000001 to 0.0001 *M*. Activity was inhibited at all levels when susceptible (S) fish were exposed. The brain fractions from S fish consistently retained more insecticide than R fish when exposed to these concentrations: 75 ppb DDT (5 hr); 2 ppb ^{14}C-endrin (6 hr); 150 ppb ^{14}C-aldrin (4 hr); and 25 ppb ^{14}C-dieldrin (4 hr). There is an effective cell membrane and brain barrier in the R fish that is less effective in the S population. In conjunction with the brain barrier, there is an apparent insensitivity to organochlorine insecticides at the site of action which is more effective in R fish than in S fish. Physical factors such as increased tissue and body fat and apparent structural membrane changes in R fish are contributory to resistance.

75-1437. Vos, J. G.; Moore, J. A.; Zinkl, J. G. (Natl. Inst. Environ. Health Sci., Res. Triangle Park, NC 27709). **Toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in C57B1/6 mice.** *Toxicol. Appl. Pharmacol. 29: 229-241; 1974.* (20 references)

Three-month-old male C57B1/6 mice were given single oral doses of 0, 100, 150, or 200 μg/kg of 2,3,7,8-tertrachlorodibenzo-*p*-dioxin (TCDD)/kg. The LD50 was 114 μg/kg. In mice that died, depletion of the thymus and spleen were consistently found and edema and terminal hemorrhages occurred frequently. In a second experiment, 4-month-old mice were dosed po with 0, 0.2, 1.0, 5.0 or 25 μg/kg, once a week for 2 or 6 weeks. Some deaths and growth retardation occurred in the 25 μg/kg dose group. Significantly increased liver

and decreased thymus weights were found in the 1, 5 and 25 μg/kg dose groups. Total neutrophils were increased significantly, whereas hemoglobin values and mean corpuscular hemoglobin concentrations were decreased significantly after 6 doses of 25 μg/kg. Total serum protein and α-, β-, and γ-globulins were significantly decreased. TCDD was porphyrogenic. The hepatic porphyria was probably associated with liver damage. Degenerative and necrotic changes in the liver were essentially centrilobular and were accompanied by cellular infiltrates and ceroid pigment deposition. Proliferation of bile duct and bile duct epithelial cells occurred. Lipid accumulation was centrilobularly localized in the mice receiving 0.2 μg/kg, was more pronounced in the mice of the intermediate dose levels, and involved hepatocytes throughout the lobule in the 25 μg/kg dose group. (Author abstract by permission)

75-1438. Guarino, A. M.; Pritchard, J. B.; Anderson, J. B.; Rall, D. P. (Lab. Toxicol., Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD 20014). **Tissue distribution of [^{14}C] DDT in the lobster after administration via intravascular or oral routes or after exposure from ambient sea water.** *Toxicol. Appl. Pharmacol. 29: 277-288; 1974.* (11 references)

A pharmacokinetic approach to studying the fate and distribution of [^{14}C]DDT was employed using the lobster, a species of obvious economic importance. The radioactive pesticide was administered by 3 different routes: intravascular, oral and by exposure from the ambient water. After intravascular administration there was very rapid removal of [^{14}C]DDT from the plasma accompanied by a strikingly persistent increase in the amount of radioactivity in the hepatopancreas. Most (> 90%) of the radioactivity in this organ was shown by TLC to be the parent pesticide. Seven days after injection of [^{14}C]DDT approximately 90% of the administered radioactivity was found in the hepatopancreas and the concentrations in this organ decreased with a $t_{½}$ of 46 days. One month after treatment with 0.1 mg/kg of [^{14}C]DDT, the only other organs which contained more than 1% of the administered dose were egg masses and muscle. When the pesticide was administered to the lobster from ambient water or from food, the hepatopancreatic compartment again dominated, with more than 90% of the absorbed dose occurring in this organ 7 days after treatment. Studies conducted of residue levels in untreated lobsters indicated that the egg masses contained the largest concentration of total DDT metabolites (1 ppm). The hepatopancreas contained about 0.4 ppm while the carcass (muscle) contained about 0.1 ppm. These distribution studies suggest that while the lobster may protect itself from DDT toxicity by sequestering the pesticide in the hepatopancreas and in egg masses, bioconcentration in these tissues could be hazardous to species consuming these parts of the lobster. (Author abstract by permission)

75-1439. Pekas, J. C. (U.S. Dep. Agric., Agric. Res. Serv., Metab. Radiat. Res. Lab., Fargo, ND 58102). **Naphthol metabolism: glucuronide conjugation and transport by the rat intestine in vitro.** *Toxicol. Appl. Pharmacol. 29: 404-419; 1974.* (15 references)

[^{14}C]Naphthol was converted to water-soluble ^{14}C-labeled metabolites by everted sacs of rat small intestine. The extent of conversion of metabolites and release to the bathing medium was greater for sacs from cranial (80%) than for sacs from caudal (60%) small intestine. The concentration of metabolites was always greater in serosal than in mucosal fluids. This concentration gradient increased from the cranial to the caudal end of the intestine. Net ^{14}C transport to the serosal fluid was entirely as metabolites and was associated with the volume of water transported. The principal metabolites were glucuronide conjugates of naphthol which were hydrolyzed by β-D-glucuronidase. Naphthyl-β-D-glucuronide appeared to be the principal metabolite in the recovered mucosal and serosal fluids and tissues. The quantities of metabolites synthesized by sacs from the various regions of the intestine were not notably different when either pH 7.4 or pH 6.5 medium was used. The tissue concentration of ^{14}C and quantity of metabolite transferred to serosal fluid in the pH 7.4 and pH 6.5 media were slightly different. The mechanism for the apparent uphill transport of metabolites was not apparent. The results have been compared to those using carbaryl and provide greater insight into the intestinal metabolism of this naphthol-containing pesticide. (Author abstract by permission)

75-1440. Eroschenko, V. P.; Wilson, W. O. (Dep. Biol. Sci., Univ. Idaho, Moscow, ID 83843). **Photoperiods and age as factors modifying the effects of Kepone in Japanese quail.** *Toxicol. Appl. Pharmacol. 29: 329-339; 1974.* (3 references)

Immature Japanese quail (*Coturnix coturnix japonica*) of both sexes fed 200 ppm of the insecticide Kepone (chlordecone), when exposed to 16 hr of light per day, had significantly enlarged reproductive organs, livers, and adrenal glands. After a reduction in the photoperiod high mortality occurred in only the birds previously fed Kepone. Administration of Kepone to adult females while egg-laying did not affect the weight of reproductive organs or livers. In adult males of the same age testes and livers were greatly enlarged. In both sexes of adult birds, the adrenals were enlarged. Subsequent exposure to reduced photoperiod of the Kepone-fed adult quails of both sexes produced no mortality. In males fed Kepone, testicular regression was recorded much earlier than in controls. Previous ingestion of Kepone affected neither the cessation of egg-laying nor the regression of the ovary. Kepone exerted its effects primarily on the immature quail of both sexes and to a lesser degree on adults. An estrogenic action of Kepone was indicated by increased oviduct weight of young females on a photoperiod of 6 hr light and 18 hr dark

per day. In adult males the major estrogenic effect was increased liver weight. (Author abstract by permission)

75-1441. La Rocca, P. T.; Carlson, G. P.; Fuller, G. C. (Dep. Pharmacol. Toxicol., Coll. Pharm., Univ. Rhode Island, Kingston, RI 02881). **Relationship between sulfhydryl reactivity and toxicity of vinyl sulfone molluscicidal agents.** *Toxicol. Appl. Pharmacol. 31(2): 222-232; 1975.* (15 references)

Vinyl sulfone was tested to determine (a) sulfhydryl reactivity, ascertained by monitoring the color-forming reaction between 5,5′-dithiobis(2-nitrobenzoic acid) and cysteine; (b) inhibition of malic dehydrogenase; (c) inhibition of in vitro endogenous respiration in the snail *Australorbis glabratus*; (d) the in vitro effect on endogenous respiration and pyruvate metabolism of liver and brain of albino rats; and (e) pyruvate metabolism in liver, kidney, and brain homogenates of male rats treated in vivo with vinyl sulfone. The effects of sublethal doses of vinyl sulfone on albino rats were also investigated. A series of vinyl sulfone molluscicidal agents was shown to exhibit a positive correlation between sulfhydryl reactivity and molluscicidal potency. High concentrations ($> 2mM$) of vinyl sulfone were required to inhibit the activity of malic dehydrogenase. No effect on endogenous respiration was observed in snails. Similarly, no effect on endogenous respiration or pyruvate metabolism was observed in rats. An ip LD50 of 3 mg/kg vinyl sulfone was found in female rats. A sublethal dose of vinyl sulfone increased blood urea nitrogen and decreased urinary output while producing no effect on SGOT and SGPT activity or on hematocrit values in rats. These data indicate that the toxicity of vinyl sulfone is not related to the inhibition of endogenous respiration in either snails or rats. The toxicity of sublethal doses of vinyl sulfone was postulated to be caused by impairment of normal renal function. (Author abstract by permission)

75-1442. Herbst, M.; Weisse, I.; Koellmer, H. (Dep. Exp. Pathol. Toxicol., C. H. Boehringer Sohn, Ingelheim, Germany). **A contribution to the question of the possible hepatocarcinogenic effects of lindane.** *Toxicology 4(1): 91-96; 1975.* (20 references)

A chronic 80-week study designed to test the possible hepatocarcinogenic effect of lindane was commenced on 500 24-day old NMRI mice which were fed a pulverized diet (Altromin-fortified). The treated groups received the same diet plus lindane in amounts of 12.5, 25, and 50 ppm (about 8 mg/kg). A comparison of food consumption, body weight development, and mortality in controls and treated animals did not suggest any influence of the compound. Autopsy showed no significant weight difference in the livers of non-tumor-bearing treated mice and controls. Four control mice, 8 mice from the 12.5 ppm group, 1 from the 25 ppm group, and 5 from the 50 ppm group displayed either leukemic changes or circumscribed round tumors of the liver. Microscopic investigations revealed the presence of liver neoplasms in a further 17 animals. The results of the study indicate that neither the occurrence of lymphatic leukemia, malignant hemangioendotheliomas, reticulum cell neoplasms, nor liver cell adenomas could be related to the administration of this compound. Considering the required safety margin, the highest dose in this study is about 650 times higher than the ADI for man, 0.0125 mg/kg. Thus the top dose seems sufficient to assess the carcinogenic risk to the consumer. On the basis of these practical values and in view of the results of experiments with different species, a carcinogenic effect of the compound in man seems very unlikely.

75-1443. Verschuuren, H. G.; Kroes, R.; Den Tonkelaar, E. M. (Lab. Toxicol., Natl. Inst. Public Health, Bilthoven, The Netherlands). **Short-term oral and dermal toxicity of MCPA and MCPP.** *Toxicology 3(3): 349-359; 1975.* (9 references)

The toxicity of the herbicides MCPA and MCPP was investigated in rats and rabbits. The compounds were added to the rat's diet at levels of 0, 50, 400, and 3200 ppm for 90 days. Both compounds caused growth retardation and elevated relative kidney weights at levels of 400 ppm and more. The 50 ppm dose level can be considered as a no-toxic effect level in the 90-day study. In subacute dermal studies in rabbits during 3 wk, the dosages were 0, 0.5, 1.0, and 2 g MCPA or MCPP per kg body weight. A recovery period of 2 wk followed. Both compounds caused slight to moderate erythema at all dose levels, while elasticity of the skin was decreased. In both experiments the skin returned to normal during the recovery period. Weight loss was observed at all dose levels. In the MCPA experiment high mortality and histopathological changes in the liver, kidneys, spleen, and thymus were recorded at the two highest dose levels.

75-1444. Verschuuren, H. G.; Kroes, R.; Den Tonkelaar, E. M.; Berkvens, J. M.; Van Esch, G. J. (Lab. Toxicol., Natl. Inst. Public Health, Bilthoven, The Netherlands). **Long-term toxicity and reproduction studies with metaldehyde in rats.** *Toxicology 4(1): 97-115; 1975.* (10 references)

Long-term toxicity and three generation reproduction studies were made with metaldehyde in rats. Rats received 0, 200, 1000, and 5000 ppm metaldehyde in the diet for 2 yr. The third litter of each generation was checked for possible embryotoxic or teratogenic effects. At 5000 ppm the relative liver weight and liver microsomal enzyme activity were increased. Posterior paralysis appeared in at least 50% of the females on 5000 ppm metaldehyde in all 3 generations. Some were affected at 1000 ppm, but none at 200 ppm. Histologically, a fracture or distortion of thoracic vertebrae and subsequent compression of the spinal cord was found. The onset of paralysis was related to the time of

delivery. The reproductive performance was impaired at 1000 and 5000 ppm, but not at 200 ppm. Subsequent generations did not show increased susceptibility to metaldehyde in this respect.

75-1445. Kay, K. (Mt. Sinai Sch. Med., New York, NY 10029). **Instrumentation in toxicology: assessing toxic materials and products in the body.** *Trans. N.Y. Acad. Sci. 36(6): 511-530; 1974.* (30 references)

The specific requirements in assessing toxic materials and their breakdown products in the body are to establish the concentration levels and follow the course of the metabolism, and to identify target organs and measure functional changes or tissue damage in these organs. Some instruments are available which permit aspects of toxicological assessment to be realized on intact body organs. In other cases blood may be drawn and small tissue samples from selected organs may be secured for examination. The main body effluents, urine, feces, and exhaled air, can be examined in a variety of ways. The use of specific instrumentation is presented in detail including atomic absorption spectroscopy, chromatography, x-ray diffraction, electron microscopy, electroencephalography, electromyography, x-radiography, and spirometry.

75-1446. Pesic, V.; Brankow, K.; Hadzovic, S. (Vet. Potpukovnici JNA, Vet. Fac., Sarajevo, Yugoslavia). **PH vrednosti pojedinih organi i tkiva normalne presadi i prasadi otravane organofosfornim jedinjenjem. [Values of pH of some organs and tissues of normal piglets poisoned with organophosphorus compounds.]** *Vet. Glas. 26(11): 815-818; 1972.* (9 references) (Serbo-Croatian)

Experiments carried out with piglets of Large White, Cornwall and Moravka breeds poisoned with armine (an organophosphorus compound) showed that there is no significant difference in the value of pH between normal animals and those poisoned with armine. It is concluded that armine, though an irreversible inhibitor of cholinesterase, has not changed the biochemical reactions (maturation) which usually take place in meat and other organs after slaughter.

75-1447. WHO Scientific Group on Chemical and Biochemical Methodology for the Assessment of Hazards of Pesticides for Man. (World Health Organization, Geneva, Switzerland). **Chemical and biochemical methodology for the assessment of hazards of pesticides for man.** *WHO Tech. Rep. Ser. 560: 1-27; 1975.* (48 references)

Chemical and biochemical methodology for the assessment of hazards of pesticides are reviewed in relation to their use in public health vector control programs that may result in human exposure. Specific analytical methods are outlined for chlorinated hydrocarbons, organophosphorus compounds, and carbamates. Biochemical assays are used to evaluate the degree of absorption of a pesticide in man and are indirect methods of assessing the history of exposure to a compound. The assay of human blood cholinesterase is widely and successfully used for monitoring persons exposed to anticholinesterase pesticides. Drug metabolism tests as indicators of the liver microsomal enzyme activity are of limited value as a general approach in monitoring exposure. The determination of pesticides and degradation products in insect larvae, snails, and rodents is discussed. Analytical methods for determining pesticides and their metabolites include spectrophotometry, thin-layer chromatography, and gas-liquid chromatography.

75-1448. Kolosovskaya, V. M. (Dep. Toxicol., Belorus. Res. Inst. Sanit. Hyg., USSR). **Izmenenie nekotorykh pokazateley obmena serotonina i aktivnosti fermenta kholinesterazy v usloviyakh dlitel'nogo vozdeystviya razlichnykh doz khlorofosa. [Alterations of some indices of serotonin metabolism and of cholinesterase activity under conditions of a durative effect of different doses of chlorophos.]** *Zdravoohkr. Beloruss. 17(1): 54-56; 1971.* (Russian)

Chlorophos (trichlorfon) was introduced daily into the stomach of white rats during 6 months in doses of 1, 2.5, 5, 10 and 15 mg/kg corresponding to 1/300, 1/150, 1/75, and 1/50 LD50. Animals were sacrified monthly and serotonin concentration, cholinesterase enzymatic activity in the blood, and the duodenal enterochromaffin cell state were determined. An analysis of the results obtained showed that changes of serotonin concentration in the blood and in the enterochromaffin cells occured considerably earlier and were more apparent than changes of the blood cholinesterase enzymatic activity. This finding may be used in hygienic standardization of organophosphorus pesticides.

see also 75-1274 75-1277 75-1281 75-1313 75-1324

75-1449. Tanaka, A.; Fujimoto, Y. (Inst. Pub. Health, Saitama, Kami-okubo, Urawa, Japan). **Colorimetric determination of biphenyl based on the Janovsky reaction.** *Agric. Biol. Chem. 38(9): 1575-1579; 1974.* (10 references)

The quantitatively nitrated product of biphenyl with potassium nitrate and sulfuric acid shows a characteristic red-purple color on reaction with isobutyl alcohol, acetone and alkali; this is the Janovsky reaction. The color reaction was sensitive, and the absorbance at 550 nm obeyed Beer's law at biphenyl concentrations between 2 and 40 μg in 3.5 ml of the reaction mixture. A procedure suitable for routine use is proposed. The nitro-compound derived from biphenyl was identified as 2,2′,4,4′-tetranitrobiphenyl by *Rf* on TLC, as well as by mixed melting point, IR and mass spectroscopy. (Author abstract by permission)

75-1450. Johnson, C. D.; Russell, R. L. (Div. of Biology, California Inst. Technol., Pasadena, CA 91109). **A rapid, simple radiometric assay for cholinesterase, suitable for multiple determinations.** *Anal. Biochem. 64(1): 229-238; 1975.* (11 references)

A rapid and simple radiometric assay for cholinesterase, suitable for multiple determinations, was developed based on a liquid-extraction technique. The assay selectively measures enzymatically produced (^{3}H)acetate by using two simple fixed-volume additions to a subsequently counted scintillation vial. The substrate for hydrolysis is (^{3}H)acetyl-labeled acetylcholine. After hydrolysis, the reaction is stopped by addition of a strong pH 2.5 buffer. This protonates the ^{3}H-labeled acetate produced, and allows it to be efficiently extracted into a large volume excess of toluene-based scintillation fluid added directly to the scintillation vial. Without further additions, the vial is simply counted; the ^{3}H-labeled acetate in the organic phase is counted efficiently, but the ^{3}H-labeled acetylcholine which remains in the small volume of aqueous reaction mixture does not count because the weak beta particles from the ^{3}H decay are trapped in the aqueous phase before reaching the scintillator.

75-1451. Klobucar, M.; Marcelja, L.; Slukan, V. (Pliva Pharm. Chem. Works, Zagreb, Yugoslavia). **Interferencija necistoca otapala kod analiza ostataka pesticida plinskom kromatografijom. [Interference of solvent impurities in pesticide residues analysis by gas chromatography.]** *Arh. Hig. Rada Toksikol. 25(2): 173-197; 1974.* (1 reference) (Serbo-Croatian)

Milk powder samples (used for baby food quality control) were analyzed and found to contain significant quantities of the pesticides α-BHC, lindane, aldrin, DDE, dieldrin, DDD (TDE), *o,p′*-DDT, and *p,p′*-DDT. The effect of unknown impurities present in the solvents was studied via changes in the GLC operating conditions, application of a very thin layer for TLC, addition of known amounts of active ingredient to the sample, and combination of mass spectrometry and GLC. The determination and isolation of the interfering components by combined mass spectrometry-GLC techniques was unsuccessful; however, it was shown that the concentrations of aldrin and α-BHC in almost all samples were lower than the values indicated by the other analytical methods.

75-1452. Marcelja, L.; Durrigl, A. (Anal. Sect., Pliva Pharm. Chem. Works, Zagreb, Yugoslavia). **Kvantitativno odredivanje tragova pesticida u nekim sirovinama za priredivanje djecje hrane. [Quantitative analysis of pesticide residues in some raw materials used in baby food production.]** *Arh. Hig. Rada Toksikol. 27(2): 257-263; 1974.* (Serbo-Croatian)

Direct quantitative determination of pesticides after their separation by TLC was utilized. DDT and its homologs absorb in the ultraviolet region of the spectrum and a mixture of known amounts of pesticides was analyzed using reflected UV radiation. The optimal wavelength for this analysis was determined and the dependence of the area under the recorded curve on the quantity of pesticides established. This method was not applicable for the analysis of unknown samples prepared by other procedures. A series of color reactions was attempted in order to compensate for interferences; the reflectance of spots obtained in this way was recorded. The relationship between light reflectance and quantity of test material, and reproducibility, accuracy, and sensitivity of these methods were discussed.

75-1453. Piedade, J. R.; Almeida, J. B. (Inst. Biol., Sao Paulo, Brazil). **Analise dos defensivos pirimidinicos. [Analysis of pyrimidine derivatives used as pesticides.]** *Arq. Inst. Biol. Sao Paulo 39(4): 335-336; 1972.* (4 references) (Portuguese)

It is shown that pyrimidine-based pesticides can be analyzed through the Kjeldahl nitrogen technique. Kjeldahl equivalents have been determined for dimethirimol (5-*n*-butyl-2-dimethylamino-4-hydroxy-6-methylpyrimidine) and pirimicarb (5,6-dimethyl-2-dimethylamino-4-pyrimidinyl dimethylcarbamate). The values obtained were 0.0069g for dimethirimol and 0.00595g for pirimicarb.

75-1454. Matsunaga, A. K.; Vasques, R. M. P.; Yoneda, H. (Instituto Biologico, Caixa Postal 7119, Sao Paulo, Brazil). **Metodo de separacao DDT-endosulfan em formulacoes de insecticidas. [Separation of DDT- Endosulfan in formulated products.]** *Arq. Inst. Biol. Sao Paulo 39(4): 337-338; 1972.* (9 references) (Portuguese)

A method is presented which permits the analysis of DDT-endosulfan formulations by gas-liquid chromatography. Since they cannot be analyzed directly by

simple extraction with acetone, the two compounds have approximately the same retention time. DDT is separated from endosulfan on a florisil chromatographic column. Since DDT is highly soluble in *n*-hexane, it is eluted with this solvent and the endosulfan remains adsorbed. Consequent elution of endosulfan is carried out with acetone.

75-1455. Yoneda, H.; Matsunaga, A. K.; Vasques, R. M. P. (Inst. Biol., Caixa Postal 7119–Sao Paulo, Brazil). **Metodo de separacao da formulacao sumithion-toxafeno. [Separation of toxaphene and sumithion in formulations.]** *Arq. Inst. Biol. Sao Paulo 40(2): 157-158; 1973.* (11 references) (Portuguese)

Because of the difficulty of analyzing sumithion (fenitrothion) by gas chromatography when it is present in a mixture with toxaphene, a new method was developed. Separation of the components is carried out on a florisil column with acetone in petroleum ether. The toxaphene is eluted with the first 60 ml of solvent, then the sumithion in the next 140 ml.

75-1456. Perna, A.; Cieri, B. (Ist. Speriment. Ig., Pescara, Italy). **Ricerca rapida di alcuni esteri fosforici per via enzimatica nelle carni dei pesci. [Rapid enzymatic determination of various organophosphorus compounds in fish flesh.]** *Atti Soc. Ital. Sci. Vet. 26: 460-462; 1972.* (4 references) (Italian)

A technique is described to simplify the analysis of dead fish following suspected intoxication by organophosphorus compounds. A cube of flesh 0.5 cm on a side is placed in a test tube containing 2 ml of 0.1 % bromthymol blue indicator and 2 ml of 3% acetylcholine chloride at a pH of 7.4 and incubated for one-half hour at a temperature of 37°C. If the fish was poisoned by an organophosphorus compound, at least 120 minutes are required for the indicator to turn yellow; the color change will be complete in 30 min if this is not the case.

75-1457. Duvenci, J. (Zirai Mucadele Ilac Aletleri Enst. Kim. Anal. Lab. Sefi, Ankara, Turkey). **Infra-red spektrofotometre ile Lasso E.C. isimli yabanciot oldurucu ilacta aktif madde tayini. [Infrared spectrophotometric determination of 2-chloro-2,6-dimethyl-*N*(methoxy-methyl) acetanilide, the active ingredient of Lasso E.C. weed killer.]** *Bitki Koruma Bul. 12(3): 153-159; 1972.* (3 references) (Turkish)

Infrared spectrophotometric analysis of 2-chloro-2,6-diethyl-*N*(methoxy-methyl) acetanilide (alachlor) in Lasso E.C. weed killer is carried out by partitioning a sample between water and chloroform, drying it on anhydrous Na_2SO_4, then measuring the absorption at wave number 1673 cm^{-1} using an infrared spectrophotometer, and finally calculating the percentage by reference to a standard curve which is prepared using the pure compound under the same conditions. Titration methods could not be used for this dark violet colored compound.

75-1458. Matsuoka, M. (Inst. Develop., Hokko Chem. Co., Atsugi, Kanagawa, Japan). **[Bioassay for antibiotics for use in agriculture.]** *Bunseki (Analysis) 2: 102-106; 1975.* (21 references) (Japanese)

Reports from 1965 to 1974 concerning the bioassay of antibiotics for agricultural and veterinary use and their residues are assembled. The bioassay method was based on cup or disk diffusion. The antibiotics and microorganisms used for analysis are as follows: streptomycin vs. *Bacillus subtilis* PCI 219, cycloheximide vs. *Saccharomyces cerevisiae* IMA 4942, novobiocin vs *Staphylococcus epidermidis* ATCC 12228, chloramphenicol vs. *Escherichia coli* NIHJ, griseofulvin vs. *Microsporum gypseum ATCC 14683*, esomycin vs. *Botrytis cinerea* ACI 1166, blasticidin S vs. *Bacillus cereus* IAM 1729, kasugamycin vs. *Pseudomonas fluorescens*, polyoxin vs. *Pellicularia filamentosa* f. *sasakii* ACI 1134 or *Alternaria mali* ACI 1157, and validamycin vs. *Pellicularia filamentosa* f. *sasakii*. For residue analysis, the residue is extracted with water and it is necessary to remove other water soluble materials. The antibiotic–microorganism combinations used for residue analysis differ from those used for formulation analysis. Detailed methods for the respective antibiotics in specific agricultural crops are described.

75-1459. Tibenska, M.; Horacek, J. (Natl. Hyg. Stn., Bratislava, Czechoslovakia.) **Prispevok k stanoveniu herbicidu 2-methyl-4-chlorfenoxyoctovej kyseliny vo vodach. [A contribution to the estimation of the herbicide 2-methyl-4-chlorophenoxyacetic acid (MCPA) in water.]** *Cesk. Hyg. 18(1): 27-32; 1973.* (14 references) (Czech)

The colorimetric estimation of MCPA with chromotropic acid showed that it is advisable to use a vacuum rotary evaporator rather than an infrared lamp for the evaporation of water based solutions. Advantages include no losses of MCPA, shorter time of evaporation, and a recovery range of MCPA from 96 to 100%. A new way of preparing the reagent is carried out by mixing 1 part of a 10% aqueous solution of the sodium salt of chromotropic acid with 99 parts of concentrated sulfuric acid. The stock solution should be prepared immediately before using and cannot be stored longer than 24 hours. The blank sample prepared in this way is minimally colored and the calibration curve of MCPA is easily reproducible for experiments using a single lot of chromotropic acid.

75-1460. Zawadzka, H.; Elbanowska, H.; Adamczewska, M. (Inst. Communal Hyg., 60-613 Poznan, Poland). **Oznaczanie herbicydow 2,4-D, dalaponu, MCPA, 2,4-DP, MCPP, DNOC, DNBP i TCA w wodzie isciewkach**

metoda chromatografii cienkowarstwowej. [Determination of herbicides 2,4-D, dalapon, MCPA, 2,4-DP, MCPP, DNOC, DNBP and TCA in water and sewage by thin-layer chromatography.] *Chem. Anal. (Warsaw) 18: 943-949; 1973.* (6 references) (Polish)

The isolation, separation, and determination of the herbicides 2,4-D, dalapon, MCPA, 2,4-DP, MCPP, DNOC, DNBP, and TCA in water and sewage by thin-layer chromatography are discussed. With regard to isolation, the method established by Bogacka and Taylor was found to be well adapted for the extraction of 2,4-D, MCPA, 2,4-DP, and MCPP from water. A chromatographic method of determining TCA as well as 2,4-D and dalapon when present simultaneously has been proposed. Optimal parameters for the chromatographic determination of the remaining herbicides are defined. Detectabilities of the methods are: 0.2 μm for 2,4-D, MCPA, 2,4-DP, MCPP, DNOC, and DNBP; 0.5 μm for dalapon; and 1 μm for TCA.

75-1461. Gertig, H.; Nowaczyk, W.; Bolewski, K. (Inst. Biol.-Farm., Akad. Med., Poznan, Poland). **Spektrofotometryczne oznaczanie wystepujacych obok siebie *p,p'*-DDT, *p,p'*-DDE i *p,p'*-DDD w roztworze cykloheksanu. [Spectrophotometric determination of coexisting *p,p'*-DDT, *p,p'*-DDE, and *p,p'*-DDD in cyclohexane solutions.]** *Chem. Anal. (Warsaw) 18(6): 1123-1128; 1973.* (8 references) (Polish)

Spectrophotometric curves were measured for DDT, DDE, and DDD (TDE) separately and for their mixtures in cyclohexane solutions. This system obeyed the sorption additivity law. Wavelengths used were 231 nm, 237 nm, and 255 nm, and absorption coefficients were determined. The absorption of mixtures of these compounds was measured and on the basis of the third absorption law, the concentration was determined. Student's test was used to confirm that the assay method could be applied for analytical purposes. Quantitative determination of DDT, DDE and DDD in butter was carried out in this way.

75-1462. Morse, C. S. (Arizona Community Studies Pesticide Project, Univ. Arizona, Tucson, AZ). **Interconversion of cholinesterase measurements by the ΔpH method of Michel and the DTNB method of Garry and Routh.** *Clin. Toxicol. 7(4):389-393; 1974.* (4 references)

There are 2 methods commonly used to measure the depression of plasma and erythrocyte cholinesterase activity as an indication of excessive absorption of organophosphate and carbamate pesticides. These methods are the ChE-tel test of Garry and Routh, which has the advantage when speed is essential such as in cases of emergency poisoning; and the Michel method, which is convenient when a large number of cholinesterase determinations are to be made at one sitting, as in worker surveillance. Because medical personnel and industrial hygienists may need to evaluate results from both methods, a simple mathematical scheme for interconversion of test values has been devised. The conversion equation for serum is: ChE-tel units = 91.3 x Michel ΔpH units, and the inverse: Michel ΔpH units = 0.0108 ChE-tel units. For red cell cholinesterase the relation is defined: AChE-tel units = 469 Michel ΔpH units, and the inverse: Michel ΔpH units = 0.00213 AChE-tel units.

75-1463. Popova, M.; Terziev, G.; Russev, P. (Dep. Forensic Med., I.P. Pavlov Fac. Med., Plovdiv, Bulgaria). **Sur la separation du dimecron d'avec les autres insecticides par la methode de chromatographie a gaz. [Separation of dimecron from other insecticides by gas chromatographic method.]** *Folia Med. (Plovdiv) 15(5): 281-284; 1973.* (11 references) (French)

A method is described for the separation of dimecron (phosphamidon) from other insecticides, such as rogor (dimethoate), phosdrin, and parathion, and for its quantitative determination in technical preparations and in cadaver materials by gas chromatography with a phosphorus-specific detector. The separation was best on a glass column, applying an evaporator temperature of 240°C, a column temperature of 180-220°C, a temperature increment of 10°C/min, a detector temperature of 240°C, and a carrier gas pressure of 2 kg/cm^2. The sensitivity of the quantitative determination on the basis of absolute calibration is 0.0001 μg.

75-1464. Malygina, Ye. F.; Tsendrovskaya, V. A. (All-Union Sci. Res. Inst. Hyg. Toxicol. Pesticides, Polymers, and Plastic Materials, Kiev, USSR). **Opredeleniye pentakhlorfenolyata natriya v atmosfernom vozdukhe metodom tonkosloynoy khromatografii. [Determination of sodium pentachlorophenolate in atmospheric air by thin-layer chromatography.]** *Gig. Sanit. 39(3): 71-72; 1974.* (1 reference) (Russian)

A thin-layer chromatographic method for the determination of sodium pentachlorophenolate in air is described. Following absorption in ethanol at a flow rate of 2 liter/min, the sample is transferred to a silica gel layer. Sodium pentachlorophenolate is visualized by means of silver nitrate in ammoniacal solution containing acetone in UV light, using a 3:2 ratio of dioxane and methanol as an eluent. The determination is based on the complex-forming reaction of sodium pentachlorophenolate with silver. The pesticide appears on the chromatogram in the form of dark gray spots. The color of the complex is stable for 2 to 3 days. The sensitivity of this method is 0.7 μg.

75-1465. Givran, V. (Cent. Chem., Minist. Educ. Invatamintului, Timisoara, Romania). **Consideratii asupra determinarii spectrofotometrice directe in ultraviolet a unor pesticide asociate. [Considerations in the direct spectrophotometric assessment in the UV range of some**

pesticide combinations.] *Igiena 22(2): 111-115; 1973.* (13 references) (Romanian)

A direct spectrophotometric method is presented for the determination of 2,4-D acid in the ultraviolet range between 250 and 350 nm, in the presence of Rogor (dimethoate) and Dipterex (trichlorfon), in a methyl alcohol medium, and for a wide range of concentrations. It was found that the relation between absorption and concentration at a wavelength of 282 nm did not undergo changes dependent on time (up to 2 hours following sample preparation). The presence of Rogor and Dipterex in samples under the experimental conditions does not influence spectrophotometric determination.

75-1466. Khan, S. U.; Purkayastha, R. (Chem. Biol. Res. Inst., Agric. Canada, Ottawa, Ontario, Canada). **Application of a thermionic detector in the analysis of *s*-triazine herbicides.** *J. Agric. Food Chem. 23(2): 311-314; 1975.* (8 references)

The response characteristics and retention times of seven *s*-triazine herbicides have been studied using a thermionic detector fitted with a cesium bromide (CsBr) tip and various gas chromatographic columns such as OV-17, SE-30, Reoplex-400, and Carbowax 20M. Thermionic detection using the Carbowax 20M column gave sensitivities comparable to those by Coulson electrolytic conductivity detector. Recoveries of atrazine added to water at 0.02, 0.05, and 2.0 ppm were between 92 and 122%; for corn at 0.1, 0.2, and 1.0 ppm, between 101 and 110%; and for soil at 0.23, 0.46, and 1.1 ppm, between 88 and 101%. The detector has also been applied to an evaluation of some extraction procedures and extracting solvents for atrazine residue in a field treated soil. (Author abstract by permission)

75-1467. Wong, L.; Fisher, F. M.* (Biol. Dep., Rice Univ., Houston, TX 77001). **Determination of carbofuran and its toxic metabolites in animal tissue by gas chromatography of their *N*-trifluoroacetyl derivatives.** *J. Agric. Food Chem. 23(2): 315-318; 1975.* (8 references)

A GLC procedure is described for the analysis of carbofuran and its toxic metabolites (3-hydroxycarbofuran and 3-ketocarbofuran) in animal samples. The residues are extracted in 0.25 *N* HCl, refluxed 1 hr at 100°, and partitioned into methylene chloride. Sample cleanup includes chromatography on activated Florisil columns and partition between 9:1 acetonitrile-water and petroleum ether. The carbamate residues are measured by GLC with electron capture detection as *N*-trifluoroacetyl derivatives. Recoveries averaged 84.2% for carbofuran, 83.8% for 3-hydroxycarbofuran, and 72.8% for 3-ketocarbofuran; minimum sensitivities are approximately 0.5, 0.005, and 0.07 ppm, respectively. (Author abstract by permission)

75-1468. Ivey, M. C.; Mann, H. D. (U.S. Livestock Insects Lab., U.S. Dep. Agric., Kerrville, TX 78028). **Gas-liquid chromatographic determination of ethion, ethion monooxon, and ethion dioxon in tissues of turkeys and cattle.** *J. Agric. Food Chem. 23(2): 319-321; 1975.* (7 references)

A gas chromatograph equipped with a flame photometric detector provided a highly sensitive method of determining residues of ethion (*S,S'*-methylene *O,O,O',O'*-tetraethyl phosphorodithioate), ethion monooxon (*O,O*-diethyl *S*-(mercaptomethyl) phosphorothioate *S*-ester with *O,O*-diethyl phosphorodithioate), and ethion dioxon (*S,S'*-methylene *O,O,O',O'*-tetraethyl phosphorothioate) in avian and animal tissues. With extraction and cleanup, 0.002 ppm of ethion, 0.002 ppm of ethion monooxon, and 0.005 ppm of ethion dioxon could be detected. Recoveries in tissues of turkeys and cattle were 79-107% for ethion and ethion monooxon and 76-98% for ethion dioxon. (Author abstract by permission)

75-1469. Greenhalgh, R.; Kovacicova, J. (Chem. Biol. Res. Inst., Agric. Canada, Ottawa, Ontario, Canada). **A chemical confirmatory test for organophosphorus and carbamate insecticides and triazine and urea herbicides with reactive NH moieties.** *J. Agric. Food Chem. 23(2): 325-329; 1975.* (15 references)

The effects of different solvents and temperatures on the base-catalyzed reaction of *O,O*-diethyl *N*-methyl phosphoramidothioate and methyl iodide were studied. The *N*-methyl derivative was obtained in high yield in dimethyl sulfoxide at 50° for 10 min with sodium hydride as the base. This alkylation procedure was adapted to the microgram level and applied to 16 insecticides and herbicides which have an NH or NH_2 moiety. With carbamates, the best yields were obtained when the reaction was carried out at room temperature. The alkylated derivatives of organophosphorus and carbamate insecticides and triazine and urea herbicides all had better GC characteristics than the parent compounds. Thus, the alkylureas readily chromatographed on SE-30/QF-1 under conditions where the parent compounds decomposed. On this column, the alkylated phosphoramidothioates and carbamates also had shorter retention times. Alkylation of crude extracts of soil, plants, and blood plasma containing herbicides and insecticides illustrated the ability of the sodium hydride-methyl iodide-dimethyl sulfoxide procedure to confirm the identity of residues at the sub parts per million level. (Author abstract by permission)

75-1470. Brown, M. J. (Agric. Canada, Res. Stn., Vancouver, British Columbia, Canada). **Improved determination of residues of phorate and its principal metabolites.** *J. Agric. Food Chem. 23(2): 334-335; 1975.* (5 references)

Improvements in methodology for the determination of residues of phorate and its three principal metabolites are described. These refinements include chilling

the sample during extraction to prevent emulsion formation, using a microcolumn cleanup procedure resulting in economy of time and materials, and evaporating under nitrogen to preclude oxidation of phorate. (Author abstract by permission)

75-1471. Newsome, W. H. (Dep. Anim. Sci., Univ. Georgia Coastal Plain Stn., Tifton, GA 31794). **A method for the determination of ethylenethiuram monosulfide on food crops.** *J. Agric. Food Chem. 23(2): 348-350; 1975.* (5 references)

A method was developed for the determination of ethylenethiuram monosulfide on food crops by gas-liquid chromatography. The procedure involves extraction with toluene followed by acid-base cleanup and yielded recoveries generally greater than 90%. The minimum detectable limit was 0.01 ppm. (Author abstract by permission)

75-1472. Suzuki, M.; Yamato, Y.; Watanabe, T. (Kitakyushu Munic. Inst. Environ. Health Sci., Tobataku, Kitakyushu, Japan). **Gas chromatographic resolution of organochlorine insecticides on OV-1/OV-17, OV-210/OV 17, and OV-225/OV-17 mixed phase systems.** *J. Assoc. Offic. Anal. Chem. 58(2): 297-300; 1975.* (17 references)

The resolution of organochlorine insecticides and their related compounds on OV-1/OV-17, OV-210/OV-17, and OV-225/OV-17 mixed phase column systems was investigated. Four BHC isomers and *p,p'*-DDT, its isomer, and their metabolites (except *p,p'*-DDE and *o,p'*-DDE) were resolved on the OV-210/OV-17 system. Heptachlor and heptachlor epoxide were separated on 5% OV-1/2% OV-17 or 5% OV-225/2% OV-17. Resolution of aldrin, dieldrin, and endrin was obtained on 5% OV-210/2% OV-17. *p,p'*-DDE and *o,p'*-DDE could not be separated by any of the systems studied. (Author abstract by permission)

75-1473. Lawrence, J. F. (Food Res. Lab., Health Protect. Br., Dep. Natl. Health Welfare, Tunney's Pasture, Ottawa, Ontario, Canada). **Effect of some operating parameters on the response of the Coulson conductivity detector.** *J. Chromatogr. 87: 333-339; 1973.* (4 references)

The variation in response with changes in operating conditions of the Coulson conductivity detector to some triazine herbicides was examined. Parameters such as water and hydrogen flow-rates as well as pyrolysis furnace temperature were investigated. Maximum response was obtained with equal flow-rates in the mixing chamber and siphon arm. However, tailing of peaks increased at slow water flow-rates. Hydrogen flow-rates of 30-70 ml/min produced the highest response. Sensitivity increased with increasing furnace temperature. (Author abstract by permission)

75-1474. Gosselin, C.; Martin, G. B.; Boudreau, A. (Dep. Vivres, Fac. Sci. Agric. Aliment., Univ. Laval, Quebec, Canada). **Contamination problems with ^{63}Ni electron capture detectors.** *J. Chromatogr. 90: 113-118; 1974.* (5 references)

A study was performed on two ^{63}Ni electron capture detectors operating in d.c. mode. Several parameters have been examined. It appeared that the temperature difference between the detector and the column is important. Scavenger gas temperature and detector isolation are not less important. Furthermore the detector polarization switch must always be off when no chromatogram is being run. These modifications help to keep the detector sensitivity much longer, with a consequent reduced number of washings. (Author abstract by permission)

75-1475. Narayanaswami, K.; Moirtra, B.; Kotangle, R. S.; Bami, H. L. (Cent. Forensic Sci. Lab., New Delhi, India). **Thin-layer chromatography in the detection of poisoning by pesticides.** *J. Chromatogr. 95: 181-188; 1974.* (29 references)

The relative efficiencies of various chromogenic reagents used in thin-layer chromatography (TLC) for the detection of commonly used pesticides in India have been evaluated. Combined diagnostic techniques using two sets of chromogenic reagents on a single TLC plate are suggested for the detection of certain organochlorine and organophosphorus pesticides. (Author abstract by permission)

75-1476. Wolkoff, A. W.; Larose, R. H. (Dep. Environ., Canada Cent. Inland Waters, Burlington, Ontario, Canada). **A highly sensitive technique for the liquid chromatographic analysis of phenols and other environmental pollutants.** *J. Chromatogr. 99: 731-743; 1974.* (33 references)

This paper describes a highly sensitive technique for the analysis of phenols and other environmental pollutants by high-speed liquid chromatography. The system utilizes a reaction detector whereby cerium(IV) sulfate is allowed to react with phenols in the column effluent, and the cerium(III) thus produced is detected by fluorescence spectroscopy. The detector shows good linearity in the region 10–230 ppb of phenol, and combined with suitable concentration techniques, the lower limit of detection of phenols in environmental samples was shown to be about 0.4 ppb. Roughly 25% of available pesticides contain a removable phenol moiety. (Author abstract by permission, supplemented)

75-1477. Musty, P. R.; Nickless, G. (Dep. Inorganic Chem., Sch. Chem., The University, Bristol, England). **The extraction and recovery of chlorinated insecticides and polychlorinated biphenyls from water porous polyurethane foams.** *J. Chromatogr. 100: 83-93; 1974.* (8 references)

Porous polyurethane foams were used for the extraction and recovery of chlorinated insecticides and polychlorinated biphenyls (PCBs) from water. One liter of tap water was doped at the ppb level and elution of the adsorbed compounds was complete with 50 ml of acetone and 100 ml of *n*-hexane. Quantitative recoveries were obtained for the thirteen insecticides used, at a water flow-rate of 100 ml/min. The recoveries for PCBs were 40–99% at 100 ml/min. The amount of methylene blue adsorbed from aqueous solution by the foams was correlated with the efficiencies of six foams, of different surface areas and bulk densities, for adsorbing chlorinated insecticides and PCBs from water. (Author abstract by permission)

75-1478. Kovac, J.; Markova, V.; Kralik, P. (Res. Inst. Agrochem. Technol., Bratislava-Predmestie, Czechoslovakia). **Gas chromatographic determination of cholinesterase inhibition.** *J. Chromatogr. 100: 171-174; 1974.* (19 references)

A new enzymatic technique for toxicological study of the inhibition of cholinesterase activity has been developed. The amount of 2,4,5-trichlorophenyl acetate (used as substrate for the esterase) and/or of 2,4,5-trichlorophenol (the product of enzymatic hydrolysis) is determined by gas chromatography, with use of an electron-capture detector selective for organochlorine compounds. This method makes possible the use of small volumes (0.1 ml) to enhance the sensitivity of trace analysis for various pesticides in toxicological work. (Author abstract by permission)

75-1479. Lawrence, J. F.; Laver, G. W. (Food Res. Lab., Health Welfare Canada, Tunney's Pasture, Ottawa, Ontario, Canada). **Analysis of triazine herbicides by combined thin-layer chromatography and fluorimetry.** *J. Chromatogr. 100: 175-179; 1974.* (13 references)

The analysis of a number of *s*-triazine herbicides by combined thin-layer chromatography and fluorimetry is investigated. The method involves hydrolysis of the triazines in 1 *N* HCl to yield cyanuric acid and the corresponding alkylamines. The amines are coupled with Dns chloride (5-dimethylaminonaphthalene-1-sulfonyl chloride) to yield highly fluorescent compounds which are separable by thin-layer chromatography. The derivatives are quantitatively evaluated by fluorimetry after removal of the spots from the plate. A variety of food samples was successfully analyzed at concentrations as low as 0.05 ppm. The method also proves useful for the confirmation of triazines which have been analyzed by other techniques, such as gas chromatography. (Author abstract by permission)

75-1480. Mendoza, C. E. (Food Drug Directorate, Dep. Natl. Health Welfare, Ottawa, Ontario, Canada). **Note on the significant difference in gas-liquid chromatographic responses to insecticides injected at fast and slow rates.** *J. Chromatogr. Sci. 9(12): 753-754; 1971.* (6 references)

The effects of injection rates on the gas-liquid chromatographic (GLC) response to Dyrene, aldrin, *p,p'*-DDT, malathion, and parathion were investigated using three electron capture detector foils. The GLC responses were significantly higher when the insecticides were injected at a slow rate than when they were injected at a fast rate. The pesticide concentrations, detector foils, and detector temperatures did not affect the results. The difference in response is apparently due to changes in size of the pesticide band moving through the column. Thus an automatic injector or the use of an EC ionization detector should improve the consistency of results.

75-1481. Deshmukh, S. N.; Singh, J. (Dept. Entomol., Punjab Agr. Univ., Ludhiana, India). **A single-step extraction and clean-up method for estimation of carbaryl in fruits and vegetables.** *J. Food Sci. Technol. (Mysore) 10(4): 194-195; 1973.* (4 references)

Carbaryl is extracted from the desired substrate with methylene chloride and the extract is passed through a Florisil column activated at 130°C for 24 hr. The insecticide is hydrolyzed with methanolic alkali and the intensity of the color developed by adding *p*-nitrobenzene diazonium fluoborate is measured at 470 nm. This method worked well with 9 substrates; okra, brinjal, cabbage, cauliflower, tomato, guava, potato, apple, and peach. The sensitivity, calculated by the method given by Bates, was found to be 0.03 ppm. Although this method is quicker than earlier methods, it is not suitable for highly colored substrates such as maize, spinach, orange, etc.

75-1482. Lakshminarayana, V. (Centr. Plant Protect. Training Inst., Hyderabad, India). **Total chlorine determinaton in organochlorine insecticides.** *J. Food Sci. Technol. (Mysore) 11(1): 15-17; 1974.* (2 references)

A method for the determination of total chlorine in aldrin, endrin, dieldrin, and lindane is described. The insecticides are dechlorinated with xylene and a small amount (about 0.2 g) of sodium. The chloride is estimated by an electrometric method. This is based on the use of a reference cell in which a silver electrode is suspended in a medium containing a small but constant concentration of silver ions.

75-1483. Sato, Y.; Fukui, S.; Sugano, S. (Kanagawa Prefect. Cent. Contr. Environ. Pollut., Kanagawa, Japan). **[Analytical method for sulfur-containing organophosphorus pesticides by FPD-gas chromatography.]** *Kanagawa-Ken Koshu Eisei Gankai-Shi (Bull. Kanagawa Prefect. Publ. Health Assoc.)20: 46-47; 1974.* (Japanese)

An analytical method of determining a sulfur-containing organophosphorus pesticide (methyl parathion) was examined. The active ingredient was

extracted under weak HCl acidic conditions, and the extract was condensed. A calibration curve was prepared with standard solutions of methyl parathion at 0.05 m*M* 0.1 m*M* and 0.2 m*M*. The conditions of gas chromatography were: 1.5 m glass column packed with 10 wt % silicone DC-200 on Anchrom ABS 90/100 mesh. The temperatures of column, inlet, and FID-IA detector point were 190, 240 and 250°C; the flow rates of hydrogen and N_2 carrier were 180 and 60 mg/min. From the surface areas of the sulfur and phosphorus peaks, the concentrations of S and P (μmole/ml) were obtained. The ratio of S/P concentrations is the ratio of atoms of S and P in the molecule. The content of the compound examined in the specimen is given by the molecular weight of the compound multiplied by the concentration of phosphorus in the specimen and by 5 (condensed volume of extract, ml), divided by the weight of specimen. It was found that the peak area per phosphorus unit molar concentration was constant regardless of the kind of organophosphorus compound.

75-1484. Abramova, E. L.; Semenova, L. N. (Inst. Chem., Acad. Sci. Ukr. SSR, USSR). **Spektrofotometricheskoye opredeleniye kotorana. [Spectrophotometric determination of cotoran.]** *Khim. Sel. Khoz. 12(2): 137-139; 1974.* (2 references) (Russian)

A rapid, highly sensitive, and selective spectrophotometric method for the determination of fluometuron (cotoran) in commercial preparations, soil, and fertilizers is presented. Following repeated extraction of the sample with chloroform, the solvent is evaporated, and the resulting crystals of fluometuron are dissolved in absolute alcohol. Two absorption maxima were found at 245 and 280 nm in aqueous alcohol, methanol, and isopropanol solutions. The shape of the spectrum and the optical density are independent of the pH value of the medium. Actual spectrophotometric determinations revealed that fluometuron in a mixture with ammophos or soil persists for a long time, but it starts degrading in 10 days in mixtures with soil plus fertilizer.

75-1485. Yoshizaki, H. (Sch. Leg. Med., Fac. Med., Kumamoto Univ., Kumamoto, Japan). **[Forensic chemical studies on synthetic organic pesticides by thin layer chromatography. Part I. Thin layer chromatography of synthetic organic pesticides.]** *Kumamoto Igakkai Zasshi (Kumamoto Med. J.) 48(6): 562-572; 1974.* (13 references) (Japanese)

A method for detecting 47 species of synthetic organic pesticides of three types, organochlorine, organomercury, and organophosphorus, by TLC was examined. These pesticides are important from the viewpoints of legal and forensic chemistry. For the purposes of this test pesticides could be classified into two large categories, one which develops a purple color and the other which does not develop a purple color during TLC, using a mixture of *n*-hexane and acetone (2:1) as the developing solvent and an ethanolic solution of diphenylcarbazide as the color reagent. The group developing a purple color was further analyzed by three kinds of TLC: (a) using a 9:1 mixture of benzene and chloroform as developing solvent and dithizone in chloroform as the color reagent; (b) using a 7:2:1 mixture of cyclohexane, acetone, and chloroform as the developing solvent and dithizone in chloroform as color developer; (c) using a 21:29:50 mixture of butanol, pyridine, and ammonia with dithizone in chloroform as the color developer. This group could be identified by the respective *Rf* value and the color of the spot. The group which did not develop the purple color was further analyzed by three kinds of TLC: (d) using an 8:1:1 mixture of cyclohexane, ethanol, and acetone as developer and bromine vapor, fluorescein sodium and UV as color reagents; (e) using a 9:1 mixture of *n*-hexane and ether as developer and an ethanolic *o*-tolidine under UV as detection system; and (f) using an 8:1:1 mixture of cyclohexane, acetone, and chloroform as developer and palladium chloride in dilute hydrochloric acid as a color reagent. The pesticides could be identified by the *RF* and the color of the spot.

75-1486. Yoshizaki, H. (Sch. Leg. Med., Fac. Med., Kumamoto Univ., Kumamoto, Japan). **[Forensic chemical studies on synthetic organic pesticides by thin layer chromatography. Part II. Detection of pesticides in cadavers of rabbits poisoned by synthetic organic pesticides.]** *Kumamoto Igakkai Zasshi (Kumamoto Med. J.) 48(6): 573-581; 1974.* (7 references) (Japanese)

Male rabbits were killed by p.o. administration of 10 times the LD50 of malathion, diazinon, sanmicron, ruberon, endrin, or lindane. Immediately after death, the stomach contents, liver, brain, blood and urine (discharged and remaining in cadaver) were removed, extracted and purified. The purified specimens were treated by TLC as described in Part I, following the respective procedures for organomercury, organochlorine, and organophosphorus compounds. Malathion and diazinon could be detected by TLC with an 8:1:1 mixture of cyclohexane, acetone, and chloroform as developer and palladium chloride as the color reagent. Sanmicron and ruberon could be detected by TLC with a 2:1 mixture of *n*-hexane and acetone as developer and diphenylcarbazide as the color reagent. Endrin and lindane could be detected by TLC with a 2:1 mixture of *n*-hexane and acetone as developer and *o*-tolidine under UV as detection system. The *Rf* values of the specimen spots coincided with those of the respective authentic pesticides except for those in brain, liver, urine, and blood of rabbits given malathion. Some pesticides were not detected in some tissues or organs, for instance, endrin in the brain and lindane in the brain.

75-1487. Langone, J. J.; Van Vunakis, H. (Dep. Biochem., Brandeis Univ., Waltham, MA 02154). **Radio-**

immunoassay for dieldrin and aldrin. *Res. Commun. Chem. Pathol. Pharmacol. 10(1): 163-171; 1975.* (16 references)

The radioimmunoassay procedure used was one described previously, in which free and antibody-bound labeled hapten derivatives were separated by the double antibody technique, except that all dilutions in this study were made in buffer containing 10% horse serum. Each reaction mixture contained (^{125}I)dieldrin derivative (20,000 cpm) and 0.1 ml of rabbit antiserum diluted 1/2000. In the absence of inhibitor, approximately 2200 cpm were precipitated. When the same amount of normal rabbit serum was added insted of immune serum to determine nonspecific binding, about 300 cpm were precipitated. The specificity of the antibodies, determined with respect to several other organochlorine insecticides, indicated that most of the binding energy is directed toward the distal part of the hapten, the hexachlorobicyclic ring system. Thus, the most effective cross reacting compounds are endrin, heptachlor, and chlordane which, like aldrin and dieldrin, are derived from Diels-Alder adducts prepared from hexachlorocyclopentadiene and appropriate dienophiles. Since the specificity of antibiodies is determined by the structure of the hapten, unrelated insecticides such as carbamates and organophosphorus compounds should not cross react with the dieldrin antibodies.

75-1488. Hinojo, J. M.; Fernandez, O. A.; Fernandez, M. E. (Dep. Cana Azucar, Estac. Exper. Agric. Tucuman, Tucuman, Argentina). **Bioensayo simple para la evaluacion de linuron en el suelo. [Simple bioassay method to evaluate linuron in soil.]** *Rev. Ind. Agr. Tucuman 49(2): 43-50; 1972.* (8 references) (Spanish)

A technique for evaluating the residual concentrations of linuron, 3-(3,4-dichlorophenyl)-1-methoxy-*a*-methylurea, in the soil is presented. Non-contaminated soil was placed in Petri dishes and a range of concentrations of linuron in solution (1,2.5,5,10,15,25,50,75 mg/l) added until the soil field capacity was reached. A comparison of the dry weight of indicator plants with regard to their growth in an unknown soil enables an approximate evaluation of the concentration of the herbicide in the soil and its potential phytotoxicity. Oats and soybeans yielded the most satisfactory results.

75-1489. Vidal, P. A.; Ricciardi, A. J. (Lab. Ribeirao Preto, Inst. Adolfo Lutz, Sao Paulo Brazil). **Metodo de dosagem turbidimetrica de pequenas quantidades de DDT e de BHC, apos sua separacao e identificacao por cromatografia em camada delgada. [Turbidimetric method for the dosage of small quantities of DDT and BHC after their separation and identification through the thin-layer chromatography process.]** *Rev. Inst. Adolfo Lutz 32: 79-82; 1972.* (4 references) (Portuguese)

A method is presented for the separation and identification of small amounts of DDT and BHC by thin layer chromatography on silica gel. Diphenylamine was used as a chromogenic reagent with exposure to ultraviolet light. The two pesticides, after development, were eluted from the chromatoplates and hydrolyzed with potassium hydroxide. The chloride ion resulting was precipitated by silver nitrate and determined by turbidimetry. It was verified that there was a linear variation of absorption in relation to the amounts of pesticide present.

75-1490. Zadrozinska, J. (Natl. Inst. Hyg., Warsaw, Poland). **Zartosowamie metody enzymatyczenej w polaczanii z chromatografia cienkowarstwowa do oznaczania pozortalosci insektycydow fosforoorganiczniych w warzywach i owocach. [Enzymatic method combined with thin-layer chromatography for determination of organophosphorus insecticides in vegetables and fruits.]** *Rocz. Panstw. Zakl. Hig. 24(3): 297-308; 1973.* (7 references) (Polish)

A method was developed for determining residues of the organophosphorus compounds methyl parathion, malathion, fenitrothion, dichlorvos, fenthion, methyl demeton, dimethoate, and trichlorfon as well as their toxic metabolites in fruits and vegetables. Insecticides were extracted from the plant materials with chloroform and the extracts divided into 2 parts. One was purified with activated charcoal and the other with petroleum ether. Activated charcoal was used for extracts containing all these insecticides with the exception of trichlorfon due to its excessive adsorption. In this case petroleum ether was used. Chromatograms were developed with a mixture of benzene, acetone and methanol (90+6+4), chloroform and acetone (9+1), *n*-hexane and ethyl acetate (4+1), and ethyl acetate. The spots on chromatograms were identified by the enzymatic method directly after irradiation of the plate with UV light, or after exposing it to ammonia vapors. The enzyme source was bovine liver and beta-naphthyl acetate with the addition of fast blue salt B as substrate. Residues of all compounds except dimethoate were found in vegetables and fruits in lower amounts than proposed tolerances. The detectability, depending on the type of the compound, the type of plant material, and the method of purification of extracts, ranged from 0.01 to 1 mg/kg.

75-1491. Fitak, B.; Spirydonow, E., Zieba, B. (Dep. Food Chem., Biopharm. Inst., Acad. Med., Warsaw, Poland.) **Reakcje barwne diptereksu i DDVP na bibule [Color reactions of dipterex and DDVP on filter paper.]** *Rocz. Panstw. Zakl. Hig. 25(1): 123-133; 1974.* (9 references) (Polish)

A study is reported on the possibility of using certain reagents for the detection of dipterex (trichlorfon) and DDVP (dichlorvos) by the drop method on filter paper. Reactions with fluorescein, 3,5-dinitro-

benzoic acid (DNB), or silver nitrate were used for the detection of dipterex, while pyrocatechol violet and dimethyl yellow were suitable for the detection of DDVP. DDVP could also be detected by color reactions on filter paper using DAB and DNB. Solutions of dipterex have a weakly acid reaction while DDVP solutions have a strongly acid one. With a rising concentration of DDVP the pH value decreases. Between the concentration of DDVP and the amount of 0.005 *N* NaOH used for titration, there is a rectilinear correlation. The effective acid strength of aqueous solutions of DDVP does not change for at least 3 hours.

75-1492. Zero, M. (Dep. Food Produce, Nat. Inst. Hyg., Warsaw, Poland). **Oznaczenie pozostosci herbicydow mocznikowych w wybranych warzywach i owocach. [Determination of urea herbicide residues in selected vegetables and fruits.]** *Rocz. Panstw. Zakl. Hig. 25(6): 667-672; 1974.* (22 references) (Polish)

Determination of linuron and monuron residues in potatoes and strawberries using thin layer chromatography is described. The method consists of extraction of these herbicides with chloroform followed by drying at 50°C. The dry residue is then dissolved in petroleum ether and extracted with acetonitrile. The acetonitrile is distilled off at 50°C and the residue dissolved in 5 ml of 25% acetone in petroleum ether. The sample is transferred to a chromatographic column filled with florisil. Because of impurities the maximum amount of extract which can be put on plates cannot exceed 10 g of plant material. In strawberries the *Rf* for linuron is 0.62, for monuron 0.27; in potatoes 0.68 and 0.38 respectively. Both herbicides could be detected in 0.05 mg/kg strawberries; linuron in 0.1 mg/kg and monuron in 0.2 mg/kg potatoes.

75-1493. Fitak, B.; Gwiazda, J. (Inst. Biopharm., Acad. Med., Warsaw, Poland). **Szybka metoda oznaczenia pozostalosci DDVP (Dichlorfosu) w ziarnie zboz. [A rapid method for evaluation of DDVP (dichlorvos) residues in grain.]** *Rocz. Panstw. Zakl. Hig. 25(6): 673-678; 1974.* (9 references) (Polish)

A rapid and simple method for the determination of microgram amounts of DDVP (dichlorvos) in wheat grain fumigated with this compound is described. It consists of extraction of a 25 g sample with 50 cm^3 of either methanol, chloroform, or methylene chloride in an agitator for 30 min, and, after addition of wood charcoal, for an additional 15 min. The extracts are then concentrated by evaporation at 30-40° to a volume of 15 cm^3, water added (20 ml), and again concentrated to complete evaporation of solvent. After addition of approx. 50 ml of water the DDVP is then colorimetrically determined (adaptation of Buchler's method) using resorcinol. With these solvents yields varied in the range of 83-86%; benzene gave lower yields, probably because of the necessity of using higher temperatures (up to 45°) for evaporation of the solvent. DDVP can also be determined directly in methanol extracts when the colorimetric method with resorcinol is used. However, it is important to maintain a proper ratio of methanol to other reagents – the volume of methanol cannot exceed 6 ml. The colorimetric method with resorcinol is not suited for determination of DDVP directly in acetone solution since it gives low yields (approximately 65%).

75-1494. Syrowatka, T.; Jurek, A. (Dept. Sanit. Toxicol., State Inst. Hyg., Warsaw, Poland). **Oznaczenie insektycydow fosfoorganicznych i karbaminianowych kinetycnza metoda enzymatyczna. [Determination of organic phosphorus and carbamate insecticides by the kinetic enzymatic method.]** *Rocz. Panstw. Zakl. Hig. 25(6): 695-699; 1974.* (9 references) (Polish)

A sensitive and rapid method for the determination of cholinesterase inhibitors such as organophosphorus and carbamate insecticides is described. It consists of an adaptation of Ellman's colorimetric method for determination of thiocholine with 5,5-dithiobis-2-nitrobenzoic acid (DTNB) after enzynatic hydrolysis of choline esters. Cholinesterase (0.4 ml) from lyophilized horse serum was added to different concentrations of chlorfenvinfos, dichlorvos, carbaryl, and propoxur in phosphorane buffer (pH 8). After vigorous shaking the samples were left standing, then 100 µl of DTNB solution and 20 µl of acetylcholine iodide were added. Extinction measurements were performed at 1 minute intervals in a spectrocolorimeter at 412 µm wave length. The percent inhibition of cholinesterase activity was determined in relation to controls and to concentration of inhibiting compound. The *I*50 (concentration inhibiting 50% of enzyme) was also determined. It was found that in vitro chlorfenvinfos was the strongest and carbaryl the weakest inhibitor. The *I*50 in these in vitro tests were as follows: chlorfenvinfos $4.1 \times 10^{-7} \pm 1.5 \times 10^{-7}$; dichlorvos $1.8 \times 10^{-2} \pm 0.4 \times 10^{-2}$; carbaryl 5.5X;1; propoxur $4.4 \times 10^{-1} \pm 1.1 \times 10^{-1}$.

PESTICIDES ABSTRACTS

QUARTERLY INDEX

APRIL–JUNE 1975 Vol. 8, Nos. 4-6

PREFACE

The basic design for this index was supplied by the Technical Services Division, Office of Pesticide Programs, Environmental Protection Agency. It consists of two subject indexes (concept and compound) and two author indexes (personal and corporate). Primary and secondary headings in the Concept Index appear as subordinate headings in the Compound Index, both indexes having three levels of specificity.

In the Concept Index, only general aspects are indexed under the broader terms shown below. For specific terms, the reader should consult the appropriate heading in the Concept Index, selecting the narrower term that best fits his need.

Compounds are indexed by their common names; where other compound designations are used by authors, these synonyms appear with the notation to *see* the common name. Articles referring, in general, to structural or functional chemical classes are indexed under the broader headings (e.g., organochlorines, herbicides, etc.).

Alternative controls
Analysis
Beneficial effects
Biochemical effects (*see also* Enzyme activity)
Blood/body fluids
Carcinogenesis
Cardiovascular system
Cytological effects
Digestive system
Economics
Endocrine system (*see also* Reproduction/growth)
Environmental pollution
Enzyme activity (*see also* Biochemical effects)
Excretory system
Experimental design
Factors influencing metabolism/toxicity
Immunology
Integumentary system
Laws and regulations
Metabolism (*see also* Residue degradation)
Morbidity and mortality statistics
Musculoskeletal system
Mutagenesis/teratogenesis (*see also* Cytological effects)
Nervous system
Nomenclature
Photodecomposition (*see also* Residue degradation)
Poison control centers
Prevention
Reproduction/growth (*see also* Endocrine system)
Residue degradation (*see also* Photodecomposition *and* Metabolism)
Residue dynamics
Residue removal
Residues/air
Residues/non-target organisms
Residues/food and feed
Residues/human
Residues/plants
Residues/soil
Residues/water
Respiratory system
Reticuloendothelial system
Reviews
Safety standards
Sensory system
Therapeutic use
Toxicity/non-target organisms
Toxicity/experimental animals
Toxicity/humans
Treatment of poisoning

Subject Index: Concepts

Absorption
see also Metabolism
Crustacea
DDT, 75-1147
Fish
DDT, 75-0916, 75-1146
Mercurials, 75-0916
Rabbit
Dichlozoline, 75-1145
Rat
Carbaryl, 75-0844
Lindane, 75-1367

Acid-base balance
see also Biochemical effects
Animals/experimental
Armine, 75-1446

Adrenal
Animals/experimental
Fenuron, 75-1171

Alimentary tract
see also Digestive system
Human
Paraquat, 75-0828

Amino acids/peptides/proteins
see also Biochemical effects
Animals/experimental
Carbamates, 75-0850
Crufomate, 75-0850
DCPA, 75-0892
Fenuron, 75-1171
Methyl parathion, 75-0843
Paraquat, 75-1173
Warfarin, 75-0944
Human, 75-1057
Chlordane, 75-0815
DDT, 75-0815
Lindane, 75-0815
In vitro
DDT, 75-1193
Dichlorvos, 75-0875
Dieldrin, 75-1193
Mercurials, 75-1115
Parathion, 75-1193
Respiration, cellular
Dichlorvos, 75-1093

Analysis
see also Bioassay; Chromatography; Electrometry; Enzyme assay; Spectrometry; Titration
General
Carbamates, 75-0968
Organochlorines, 75-1228
Organophosphates, 75-0968
Experimental design, 75-0987
Reviews
Arsenicals, 75-1001
Herbicides, 75-0967
Mercurials, 75-1001
Sample preparation, 75-0971 75-1231
Carbaryl, 75-1481

Analysis (cont'd)
DDE, 75-0980
DDT, 75-0980
Hexachlorobenzene, 75-0980 75-1215
Hexachlorobutadiene, 75-1215
Lindane, 75-0980
Phorate, 75-1470
Polychlorinated biphenyls 75-0980
TDE, 75-0980

Balance
Animals/experimental
DDT, 75-1165

Behavior
see also Nervous system
Animals/experimental
DDT, 75-1147, 75-1165, 75-1166 75-1208, 75-1210
DFP, 75-1176
Dieldrin, 75-1210
Fenitrothion, 75-1163, 75-1164
Parathion, 75-1210
Toxaphene, 75-1210
Human
Dioxins, 75-1306

Bile
see Blood/body fluids

Bioassay
see also Analysis 75-0970
Antibiotics, 75-1458
Benomyl, 75-0988
Fenthion, 75-1224
Linuron, 75-1488
Organochlorines, 75-1229

Biochemical effects
see also Acid-base balance; Amino acids/peptides/proteins; Carbohydrates; Cytochromes; Electrolytes; Enzyme activity; Lipids/steroids/sterols; Nucleic acids; Porphyrins; Respiration, cellular; Vitamins/coenzymes
General
Dinitrophenol, 75-0882
Hydrogen cyanide, 75-0882
Pyrethrins, 75-1372
Rotenone, 75-1372
Sodium fluoride, 75-0882
Sodium fluoroacetate, 75-0882
2,4,5-T, 75-0904
Trichlorfon, 75-1415
Animals/experimental
BHC, 75-1169
Captafol, 75-1189
DEF, 75-1169
Hexachlorobenzene, 75-1153

Biochemical effects (cont'd)
Molluscicides, 75-1441
Promecarb, 75-1365
Thiram, 75-1169
Toxaphene, 75-1281
Zineb, 75-1189
Microorganisms
Benomyl, 75-1204
Bromacil, 75-0934
Dichlorvos, 75-1199
Diphenamid, 75-0934
Fungicides, 75-1205
Malathion, 75-1199
Prometryne, 75-0934
Thiram, 75-1156
Trichlorfon, 75-1199

Biotransformation
see also Metabolism
General, 75-1429, 75-1430, 75-1431
Mercurials, 75-1358
Organophosphates, 75-0960
Phenamiphos, 75-0962
Prolan, 75-1194
R-16661, 75-0958
Annelids
Benomyl, 75-0929
Chicken
3-Chloro-*p*-toluidine, 75-1078
Fish
TFM, 75-0951
Fungi
Alachlor, 75-0893
Carbaryl, 75-1140
Helminths
Phorate, 75-1184
Human
Diazinon, 75-1056
PCP, 75-1107
In vitro
Benthiocarb, 75-1192
Biphenyl, 75-1352
Bromophos, 75-1423
Butylate, 75-1192
Cisanilide, 75-1195
Cycloate, 75-1192
Dichloroaniline, 75-1339
Dicrotophos, 75-0927
Dimethoate, 75-0927
EPTC, 75-1192
Methiocarb, 75-0885
Mexacarbate, 75-0885
Molinate, 75-1192
Nitro compounds, 75-1098
Organophosphates, 75-1120
Pebulate, 75-1192
Phosphamidon, 75-0927
Vernolate, 75-1192
Insects
Bombykol, 75-0914
Carbamates, 75-1434
DDT, 75-1434
DDT derived compounds 75-1434

Biotransformation (cont'd)
Juvenile hormones and analogs 75-0926
Organophosphates, 75-1434
Microorganisms, 75-1100, 75-1274
Alkanes, 75-0846
Benomyl, 75-1204
Carbaryl, 75-1141
2,4-D, 75-1124
DDT, 75-1141
Dioxacarb, 75-0913
Diuron, 75-1206
Dyfonate, 75-1123
Herbicides, 75-1196
Ioxynil, 75-1020
Malathion, 75-1134
Monuron, 75-1206
1-Naphthol, 75-0894
Organophosphates, 75-1119
Parathion, 75-0907, 75-1141
PCP, 75-1128
PCP derived compounds, 75-0872 75-1177
Phorate, 75-1184
Prometryne, 75-0935
Trifluralin, 75-0956
Mouse
Benthiocarb, 75-1192
Butylate, 75-1192
Cycloate, 75-1192
2,4-D, 75-1105
Endrin, 75-1061
EPTC, 75-1192
Molinate, 75-1192
PCP, 75-1107
Pebulate, 75-1192
Vernolate, 75-1192
Plants
Cyanazine, 75-0903
Fenthion, 75-1417
Heptachlor, 75-1349
Methoprene, 75-1158
Neodecanoic acid, 75-1159
Norflurazon, 75-0955
Parathion, 75-1378
Salithion, 75-0852
San 6706, 75-0955
Triphenyltin, 75-0931
Rat, 75-0852
Carbaryl, 75-1423
2-Chloro-4-acetotoluidine, 75-1078
2,4-D, 75-1105
Endrin, 75-0880
PCP, 75-1107
Pyrethrins, 75-0896
2,4,5-T, 75-1105
2,4,5-TB, 75-1106
TDE, 75-1129
Toxaphene, 75-0895
Triphenyltin, 75-0931
Ziram, 75-1362

Blood/body fluids
see also Bile; Blood cells
Animals/experimental
Dichlozoline, 75-1145
Dioxins, 75-1437
In vitro
Mercurials, 75-1427

Blood cells
see also Blood/body fluids
Animals/experimental
Atropine, 75-1390
Fenuron, 75-1171
Organophosphates, 75-1211
Pralidoxime, 75-1390
Propanil, 75-0953
Thiometon, 75-1390
Human
Arsenicals, 75-1043
MCPA, 75-1302
In vitro
Paraoxon, 75-0855

Blood pressure
see also Cardiovascular system
Animals/experimental
Acrolein, 75-0945
DFP, 75-0860

Blood vessels
see Cardiovascular system

Brain
see also Nervous system
Animals/experimental
Chlorfenvinphos, 75-1399
DDT, 75-0932
DFP, 75-1333
Leptophos, 75-1096
Milbex, 75-1143
Organophosphates, 75-1211
Phencapton, 75-1185
Human
Mercurials, 75-1000

Carbohydrates
see also Biochemical effects
Animals/experimental
DCPA, 75-0892
DDT, 75-1337
Fluorine compounds, 75-1187
M-1704, 75-0920
Mercurials, 75-1187
Norbormide, 75-0891
Organophosphates, 75-1187
In vitro
DDT, 75-0853

Carcinogenesis
General, 75-0948, 75-1200
Amitrole, 75-0818, 75-0827
Paraquat, 75-0828
Animals/experimental
Amitrole, 75-1409, 75-1410
BHC, 75-1411, 75-1412
DDT, 75-0867, 75-1411
Dioxins, 75-1344
Lindane, 75-1442
Polychlorinated biphenyls 75-0867
Human, 75-1307, 75-1407
Arsenicals, 75-0813, 75-1303
DDT, 75-0812
Lindane, 75-0812
Organophosphates, 75-0812

Cardiovascular system
see also Blood pressure; Blood vessels; Heart
Animals/experimental
Plictran, 75-0949

Catecholamines
Animals/experimental
Dieldrin, 75-0940
Paraquat, 75-1076
Trichlorfon, 75-1448
In vitro
Paraquat, 75-1067

Cell membranes
Animals/experimental
Parathion, 75-1109
In vitro
Dichlone, 75-0878
Organophosphates, 75-0888 75-0889
Microorganisms
DDT, 75-1063

Chromatography
see also Analysis
General
Dioxins, 75-0992
Heptachlor, 75-1232
Heptachlor epoxide, 75-1232
Lindane, 75-1232
Column
Carbamates, 75-1222
DDT derived compounds 75-0973
Fenitrothion, 75-1455
Maleic hydrazide, 75-0984
Organochlorines, 75-0974
Organophosphates, 75-0993 75-1222
Polychlorinated biphenyls 75-0973
Toxaphene, 75-1455
Gas-liquid, 75-0982
Atrazine, 75-0976
Bay 94337, 75-0985
Carbamates, 75-1213, 75-1220 75-1469
Carbofuran, 75-1467
DDT, 75-1454
Endosulfan, 75-1454
Ethion, 75-1468
Ethion derived compounds 75-1468
Fenitrothion derived compounds 75-0990
Formothion, 75-0978
Fungicides, 75-1220, 75-1471
Herbicides, 75-1220, 75-1469
Maleic hydrazide, 75-0984
Methoprene, 75-0983
Methyl bromide, 75-1225
Methyl parathion, 75-1483
Organochlorines, 75-1010 75-1213, 75-1217, 75-1451 75-1472, 75-1474, 75-1480
Organophosphates, 75-1213 75-1469, 75-1480
PCP, 75-0972

Chromatography (cont'd)
Phenols, 75-1476
Phorate, 75-1470
Phosphamidon, 75-1463
PLP derived compounds, 75-1223
Polychlorinated biphenyls 75-0972, 75-1217
Pyrazon, 75-0989
Pyridafenthion, 75-1226
Simazine, 75-0976
2,4,5-T, 75-1478
Triazines, 75-1466, 75-1473
Paper
Formothion, 75-0978
Thin-layer, 75-0979
Atrazine, 75-1216
Benomyl, 75-0996
BHC, 75-1489
Bombykol, 75-0914
Crimidine, 75-1219
2,4-D, 75-1460
Dalapon, 75-1460
DDT, 75-0981, 75-1216, 75-1452 75-1489
Dieldrin, 75-1216
DNBP, 75-1460
DNOC, 75-1460
2,4-DP, 75-1460
Heptachlor, 75-1234
Heptachlor epoxide, 75-1234
Lindane, 75-1216
Linuron, 75-1492
Malaoxon, 75-1230
Malathion, 75-1230
MCPA, 75-1460
MCPP, 75-1460
Mercurials, 75-1485, 75-1486
Methabenzthiazuron, 75-1221
Monuron, 75-1492
Naptalam, 75-1221
Organochlorines, 75-1475 75-1485, 75-1486
Organophosphates, 75-0994 75-1475, 75-1485, 75-1486 75-1490
PCP, 75-1464
Phencapton, 75-1227
Phenmedipham, 75-1233
Propanil, 75-0953
Pyridafenthion, 75-1226
Rotenone, 75-1221
Simazine, 75-1216
TCA, 75-1460
Thiometon, 75-1227
Thioquinox, 75-1221
Triazines, 75-1479
Warfarin, 75-1221

Chromosomes/genes
see also Cytological effects; Mutagenesis/teratogenesis
Animals/experimental
Trichlorfon, 75-1368
Zineb, 75-1368
Human
Irradiation, 75-1102
Organophosphates, 75-1046
Toxaphene, 75-0839

Chromosomes/genes (cont'd)
Zineb, 75-1049
In vitro
Aldrin, 75-1397
Azinphosmethyl, 75-0881
Phosphamidon, 75-1397
Microorganisms
Carbaryl, 75-1396

Cytochromes
see also Biochemical effects
Animals/experimental
DDT, 75-1433
Mirex, 75-1433
Synergists, 75-1433

Cytological effects
see also Chromosomes/genes; Endoplasmic reticulum; Mitochondria; Mitosis/meiosis
Animals/experimental
Dieldrin, 75-1391
Dioxins, 75-1344
Disulfoton, 75-1338
Malathion, 75-1421, 75-1424
Trichlorfon, 75-1415
Human, 75-1407
Chlordane, 75-1323

Demyelination
see also Nervous system
Animals/experimental
Leptophos, 75-1096

Digestive system
see also Alimentary tract; Liver
Animals/experimental
Bordeaux mixture, 75-0919
Carbaryl, 75-0919
DDT, 75-0919
Dieldrin, 75-0919
Mercurials, 75-0919
Toxaphene, 75-0919
In vitro
Organochlorines, 75-1405
Organophosphates, 75-1405

Distribution/storage
see also Metabolism
Birds
DDT, 75-0866
Crustacea
DDT, 75-1438
Duck
DDT, 75-1385
Dieldrin, 75-1386
Fish
DDT, 75-1146
Human
Mevinphos, 75-1044
Mouse
Amitrole, 75-1110
DDT, 75-1401, 75-1402
Dieldrin, 75-1401, 75-1402
Plants
Carboxin, 75-0791
Norflurazon, 75-0955
San 6706, 75-0955
Rat
Carbaryl, 75-0780

Distribution/storage (cont'd)
Carbofuran, 75-0780
DDT, 75-0780, 75-1161
Leptophos, 75-0780
Mirex, 75-0780
Organochlorines, 75-1313
Parathion, 75-1363
Warfarin, 75-0912

Economics, 75-1238, 75-1245
Herbicides, 75-1237, 75-1242

EEG
Human
2,4-D, 75-1325
MCPA, 75-1325

Eggshell effects
see also Reproduction/growth
Animals/experimental
DDE, 75-0849, 75-1097, 75-1148
Mirex, 75-1062, 75-1103
Animals/non-target
Organochlorines, 75-0768

Electrolytes
see also Biochemical effects
Animals/experimental
DCPA, 75-0892
DDE, 75-1066, 75-1097
DDT, 75-1126
Hexachlorobenzene, 75-1150
2,4,5-T, 75-1083
In vitro
Parathion, 75-1070
Microorganisms
Fungicides, 75-1205

Electrometry
see also Analysis
Amperometry
Organochlorines, 75-1482
Polarography
Heptachlor, 75-1232, 75-1234
Heptachlor epoxide, 75-1232 75-1234
Lindane, 75-1232

Embryo/fetus
see also Reproduction/growth
Animals/experimental
Amitrole, 75-1110
Cuprozin, 75-1144
DDT, 75-1146
Paraquat, 75-0871
Human
DDT, 75-1022
In vitro
Methiocarb, 75-0885
Mexacarbate, 75-0885

Endoplasmic reticulum
see also Cytological effects
Animals/experimental
DDT, 75-0887, 75-1371

Environmental pollution, 75-0998 75-1240, 75-1241, 75-1246
Arsenicals, 75-1001
Mercurials, 75-1000, 75-1001
Organochlorines

Environmental pollution (cont'd)
Organochlorines, 75-1264
Polychlorinated biphenyls, 75-1244

Enzyme activity
see also Biochemical effects
General, 75-1057, 75-1181, 75-1418 75-1426
Atropine, 75-1390
BHC, 75-1170
Carbaryl, 75-0877
Chlortoluron, 75-1422, 75-1425
Cuprozin, 75-1144
DDT, 75-1371, 75-1420
DFP, 75-1176
Diuron, 75-1420, 75-1422
Fluenethyl, 75-1353
Hexachlorobenzene, 75-1153 75-1375
Linuron, 75-1420, 75-1422 75-1425
Malathion, 75-0877, 75-1421 75-1424
MNFA, 75-1370
Pralidoxime, 75-1390
Pyrethrins, 75-1372
Rotenone, 75-1372
Thiometon, 75-1390
Trichlorfon, 75-1415
α-chymotrypsin
Organophosphates, 75-1120
Acetanilide hydroxylase
Propoxur, 75-1348
Acid phosphatase
Captan, 75-1414
Aliesterase
Carbamates, 75-1434
DDT, 75-1434
DDT derived compounds 75-1434
Organophosphates, 75-1434
Alkaline phosphatase
Ziram, 75-1408
5-Aminolevulinate synthase
Lindane, 75-1149
Aminopyrine *N*-demethylase
Propoxur, 75-1348
Animals/experimental
DEF, 75-1169
ATPase
Chlordecone, 75-1342
DDE, 75-1066, 75-1097, 75-1148
DDT, 75-0845, 75-1122
Dichlone, 75-0878
Dioxins, 75-1344
NS 2662, 75-1400
Carbonic anhydrase
DDE, 75-1097, 75-1148
Catalase
Amitrole, 75-0900, 75-0946
Mercurials, 75-1427
Cathepsin A
DFP, 75-0899
Cholinesterase, 75-0796, 75-1052 75-1058, 75-1168
Armine, 75-1446
Azinphosmethyl, 75-0823 75-0863

Enzyme activity (cont'd)
Benomyl, 75-0929
Carbamates, 75-0858, 75-0968 75-1321
Carbaryl, 75-1162
Carbofuran, 75-1104
Chlorfenvinphos, 75-1399
Chlorpyrifos-methyl, 75-0771
Copper, 75-1142
Coumaphos, 75-0915
DDT, 75-0851
DFP, 75-1068, 75-1333
Dichlorvos, 75-0842, 75-1081 75-1191, 75-1327
Dichlozoline, 75-1145
Dicofol, 75-1142
Diquat, 75-1077
Ethion, 75-0823
Fenitrothion, 75-0942, 75-1190
Formetanate, 75-1366
Leptophos, 75-1096
Malaoxon, 75-0905
Malathion, 75-0905, 75-1155
Methyl parathion, 75-0863
Mevinphos, 75-1044
Morfamquat, 75-1077
Naled, 75-0823, 75-0950
Organochlorines, 75-1315 75-1316
Organophosphates, 75-0817 75-0822, 75-0858, 75-0968 75-1082, 75-1116, 75-1211 75-1315, 75-1316, 75-1321
Paraoxon, 75-0905, 75-1138
Paraquat, 75-1077
Parathion, 75-0863, 75-0905 75-1080, 75-1104, 75-1109 75-1175
Phosalone, 75-0823
Pralidoxime, 75-0939
Propham, 75-1327
Soman, 75-1091
Tepa, 75-1114
Tetrachlorvinphos, 75-1191
Thio-tepa, 75-1108
Thiometon, 75-1364
Trichlorfon, 75-1117, 75-1142 75-1419, 75-1448
Cytochrome oxidase
Copper oxychloride, 75-1404
Dichlorvos, 75-1404
MCPA, 75-1404
Milbex, 75-1143
Dehydrogenase
Benomyl, 75-1204
Esterases
Carbamates, 75-1118
Organophosphates, 75-1118 75-1120
Paraoxon, 75-0855
β-Glucuronidase
DFP, 75-1099
Paraoxon, 75-1099
GOT
Tetrachlorvinphos, 75-1191
Thiram, 75-0890
Zineb, 75-0890

Enzyme activity (cont'd)
Lactic dehydrogenase, 75-1052
Hexachlorobenzene, 75-1270
Mirex, 75-1087
Mixed function oxidases, 75-0883 75-1090
Aldrin, 75-1095
BHC, 75-1169
BHC isomers, 75-0924
Carbamates, 75-1434
DDT, 75-0848, 75-0884, 75-1198 75-1201, 75-1337, 75-1433 75-1434
DDT derived compounds 75-1434
DEF, 75-1169
Dichlorvos, 75-1093
Dieldrin, 75-0848, 75-0909 75-0927
Dioxins, 75-0857
Hexachlorobenzene, 75-0884 75-1131
Lindane, 75-1406
Malathion, 75-0933
Mirex, 75-1062, 75-1433
α-Naphthyl isothiocyanate 75-0876
Organophosphates, 75-1434
Piperonyl butoxide, 75-0941 75-1179, 75-1201
Synergists, 75-1433
Thiram, 75-1169, 75-1207
p-Nitrophenol glucuronyltransferase
Dioxins, 75-1071
para-Nitrophenyl phosphatase
Organophosphates, 75-0888 75-0889
Ornithine decarboxylase
DDT, 75-1094
TDE, 75-1094
Pepsin
Organochlorines, 75-1405
Organophosphates, 75-1405
Succinate dehydrogenase
DNBP, 75-1369
Lindane, 75-1369
Milbex, 75-1143
UDP glucuronyltransferase
Dioxins, 75-0856
Urease
Dalapon, 75-1154
Paraquat, 75-1154

Enzyme assay
see also Analysis
Cholinesterase, 75-1450, 75-1462
Carbamates, 75-1494
Organophosphates, 75-1456 75-1494
2,4,5-T, 75-1478

Estrogens
see Reproduction/growth

Excretion
see also Metabolism
Chicken
DDT, 75-1125

Excretion (cont'd)
Cow
Chlorpyrifos-methyl, 75-0771
Duck
DDE, 75-0865
DDT, 75-0865
Dieldrin, 75-0865
Fish
DDT derived compounds 75-1086
Human
Mercurials, 75-1000
In vitro
DDT derived compounds 75-1069, 75-1086
Mouse
BHC isomers, 75-0943
Mercurials, 75-1388
Polychlorinated biphenyls 75-0943
Rat
DDT, 75-1161
Mercurials, 75-1388
Salithion, 75-0852
2,4,5-T, 75-1083
TDE, 75-1129

Excretory system
see also Kidney
Animals/experimental
Dioxins, 75-1376
2,4,5-T, 75-1361

Experimental design
General, 75-1445
Analysis, 75-0987
Monitoring and residues
DDT, 75-1252
Toxicology and pharmacology 75-0796, 75-0948, 75-1178 75-1447
Carbamates, 75-0858
Carbaryl, 75-0844
Mirex, 75-1343
Organophosphates, 75-0858
Propoxur, 75-1065

Factors influencing metabolism/toxicity
Adaptation
Carbamates, 75-1434, 75-1436
DDT, 75-1434, 75-1436
DDT derived compounds 75-1434
Herbicides, 75-1356
Organochlorines, 75-1347 75-1435
Organophosphates, 75-1434 75-1436
Age, 75-1312
Aldrin, 75-1072
Carbaryl, 75-1377
Chlordecone, 75-1439
Organochlorines, 75-1183
Parathion, 75-1080, 75-1175
2,4,5-T, 75-1361
Thiram, 75-1207
Biological magnification
Photodieldrin, 75-0847
Disease state
DDT, 75-1385
Paraoxon, 75-1138
Formulation
2,4-D, 75-1383
Dieldrin, 75-1386
Lindane, 75-1367
Paraquat, 75-1314
Humidity
Dichlorvos, 75-1081
Terbutryne, 75-0794
Interactions, 75-0850
Amitrole, 75-0946, 75-1409
BHC, 75-1411, 75-1412
Carbaryl, 75-1140
Copper, 75-1052, 75-1142
DDE, 75-0864
DDT, 75-0864, 75-1198, 75-1411
Dicofol, 75-1142
Formothion, 75-1052
Granosan, 75-1052
Lindane, 75-1136, 75-1149
Mercurials, 75-0946, 75-1388
Organochlorines, 75-0936
Organophosphates, 75-0936
Parathion, 75-1418
Polychlorinated biphenyls 75-0864
Salicylate, 75-0947
Trichlorfon, 75-1142
Warfarin, 75-0947
Light/radiation
Chlordecone, 75-1439
Photodieldrin, 75-1340
Molecular structure
Carbamates, 75-1118
Demeton, 75-0859
Herbicides, 75-1196
Methylmercuric chloride, 75-1132
Organophosphates, 75-0958 75-0959, 75-0961, 75-0963 75-1118
Phenylmercuric chloride, 75-1132
Pyrethrins, 75-1016
Nutritional state
Captan, 75-1414
DDT, 75-1127
Dyfonate, 75-1123
Norbormide, 75-0891
Pralidoxime, 75-0939
Warfarin, 75-0944
pH, 75-1181
Dyfonate, 75-1123
Herbicides, 75-0930
Pralidoxime, 75-0939
Route, 75-0850
Plictran, 75-0949
Season, 75-1057
Sex, 75-1312
BHC, 75-1412
Carbamates, 75-1162
Carbaryl, 75-1064, 75-1377
5-*p*-chlorophenyl silatrane 75-1167
Dichlorvos, 75-1064
Dioxins, 75-1071, 75-1376
Fospirate, 75-1079
Hexachlorobenzene, 75-1131 75-1270
Parathion, 75-1175
Trichlorfon, 75-1079
Taxon
Azinphosmethyl, 75-0863
BHC, 75-1412
BHC isomers, 75-0921
Bromophos, 75-1423
2-Chloro-4-acetotoluidine, 75-1078
3-Chloro-*p*-toluidine, 75-1078
DDT, 75-1125, 75-1193, 75-1210
Dieldrin, 75-1193, 75-1210
Endrin, 75-1061
Herbicides, 75-1196
Hexachlorobenzene, 75-0886
Lindane, 75-1149
Methyl parathion, 75-0863
Parathion, 75-0863, 75-1193 75-1210
Photodieldrin, 75-0847
Polychlorinated biphenyls 75-0886
Thio-tepa, 75-1108
Toxaphene, 75-1210
Temperature
DDT, 75-0916
Dyfonate, 75-1123
Mercurials, 75-0916
Terbutryne, 75-0794
Thyroidectomy
Amitrole, 75-1410

Feathers
see also Integument
Animals/experimental
Methyl parathion, 75-0843

Fertility/sterility
see Reproduction/growth

Gills
see Respiratory system

Glands
see also Integument
Animals/non-target
DDE, 75-0865
DDT, 75-0865
Dieldrin, 75-0865

Growth
see also Reproduction/growth
Animals/experimental
Captafol, 75-1189
Carbamates, 75-0850
Chlorpyrifos-methyl, 75-0771
Coumaphos, 75-0915
Crufomate, 75-0850
DDT, 75-0850, 75-0884
Dioxins, 75-1376
Malathion, 75-1416
Thiram, 75-0890
Trichlorfon, 75-1416
Zineb, 75-0890, 75-1189
Microorganisms
Aldrin, 75-1394
Arsenicals, 75-0954
BHC, 75-1394

Growth (cont'd)
Dalapon, 75-1154
DBCP, 75-1379
D-D, 75-1379
DDT, 75-1063, 75-1394
Dichlorvos, 75-1199
Linuron, 75-1393
Malathion, 75-1199
MNFA, 75-1370
Paraquat, 75-1154
PCP, 75-1392
Trichlorfon, 75-1199
Plants
Dichlorvos, 75-1199
Malathion, 75-1199
Trichlorfon, 75-1199

Hair/fur
see Integument

Heart
see also Cardiovascular system
Animals/non-target
Atropine, 75-1390
Pralidoxime, 75-1390
Thiometon, 75-1390

Immunology
General
Zineb, 75-1368
Animals/experimental
DDT, 75-1385
Dieldrin, 75-1386
O-Ethyl-*S*-(2-diethylaminoethyl)m-ethylthi, 75-1075
Paraoxon, 75-1075
Sarin, 75-1075
Soman, 75-1075
In vitro
DFP, 75-1384

Integument
see Feathers; Glands; Hair/fur; Skin

Kidney
see also Excretory system
Animals/experimental
Copper, 75-1142
Dicofol, 75-1142
Fenuron, 75-1171
Propanil, 75-0953
2,4,5-T, 75-0904
Trichlorfon, 75-1142
Human
Paraquat, 75-1045
In vitro
DDT, 75-0853
Parathion, 75-1070
Rat
Formetanate, 75-1366
Promecarb, 75-1365

Laws and regulations
General, 75-0835
DDT, 75-0825
Finland, 75-0826
Japan, 75-0838
Sweden, 75-0814, 75-0819
USA-EPA, 75-1241
World Health Organization, 75-1247
Yugoslavia, 75-1239

Lipids/steroids/sterols
see also Biochemical effects
Animals/experimental
DDT, 75-1084, 75-1085
DDT derived compounds 75-1152
Dieldrin, 75-1133
Dioxins, 75-1071
M-1704, 75-0920
Propoxur, 75-1348
Human
Chlordane, 75-0815
DDT, 75-0815
Dioxins, 75-1306
Lindane, 75-0815
In vitro
2,4-D, 75-0854
Dichlorvos, 75-1088
Dieldrin, 75-1133
2,4,5-T, 75-0854

Liver
see also Digestive system
Animals/experimental, 75-0796 75-1090, 75-1191, 75-1426
Amitrole, 75-1409, 75-1410
BHC, 75-1169, 75-1411, 75-1412
Carbamates, 75-0850
Chlordane, 75-1074
Chlortoluron, 75-1422, 75-1425
Crufomate, 75-0850
DDT, 75-0848, 75-0850, 75-0884 75-1127, 75-1198, 75-1337 75-1385, 75-1411, 75-1433
Dichlorvos, 75-1093
Dieldrin, 75-0848, 75-0909 75-1386
Dimethoate, 75-0841
Dioxins, 75-0857, 75-1344 75-1376, 75-1437
Diuron, 75-1422
DNBP, 75-1369
Fenuron, 75-1171
Hexachlorobenzene, 75-1150
Lindane, 75-1149, 75-1369 75-1406
Linuron, 75-1422, 75-1425
Malathion, 75-0933, 75-1421 75-1424
Mirex, 75-1062, 75-1433
Organochlorines, 75-1347
Parathion, 75-1109
Polychloropinene, 75-1172
Propanil, 75-0953
Synergists, 75-1433
2,4,5-T, 75-0904
Thiram, 75-0890, 75-1169 75-1207
Trichlorfon, 75-0841, 75-1415
Warfarin, 75-0944
Zineb, 75-0890
Human, 75-0808, 75-1058
Chloramphenicol, 75-1308
Chlordane, 75-1323
Novobiocin, 75-1308
Organophosphates, 75-1308
Paraquat, 75-1045

Liver (cont'd)
In vitro
DDT, 75-0853, 75-1201
Dichloroaniline, 75-1339
Malathion, 75-0877
Mercurials, 75-1427
α-Naphthyl isothiocyanate 75-0876
Piperonyl butoxide, 75-1201
Mixed function oxidases
Piperonyl butoxide, 75-0941
Rat
Formetanate, 75-1366
Promecarb, 75-1365

Lung
see also Respiratory system
Animals/experimental
Diquat, 75-1077
Morfamquat, 75-1077
Paraquat, 75-0862, 75-0917 75-1077
Thio-tepa, 75-1108
Zineb, 75-1189
Human
Amitrole, 75-0818
Bordeaux mixture, 75-1041
Copper sulfate, 75-1041
Paraquat, 75-0828, 75-0831 75-1045, 75-1059
Rat
Formetanate, 75-1366

Marrow
see Reticuloendothelial system

Metabolism
see also Absorption; Biotransformation; Distribution/storage; Excretion
General
Dalapon, 75-0964
DDT, 75-1433
trans-4,5-dihydroxy-4,5-dihydroaldrin, 75-0879
Herbicides, 75-1428
Methoxychlor derived compounds 75-0910
Mirex, 75-1433
Organophosphates, 75-0959
Photodieldrin, 75-0847
Pyrazon, 75-1277
Synergists, 75-1433
Dog
2,4,5-T, 75-1361
Fish, 75-1432
Human
Carbaryl, 75-1373
In vitro
Aldrin, 75-1072
Carbaryl, 75-1373
Chlordimeform, 75-1374
Phenothrin, 75-0928
Insects
Carbamates, 75-1436
Carbaryl, 75-1377
DDT, 75-1436
Organophosphates, 75-1436
Microorganisms

Metabolism (cont'd)
Aniline, 75-1359
Carbaryl, 75-1354
DDT, 75-1354
Herbicides, 75-0930
Parathion, 75-1354
Mouse
Ethylene oxide, 75-1395
Plankton/algae
Atrazine, 75-1341
Carbaryl, 75-1341
2,4-D, 75-1341
Diazinon, 75-1341
Hymexazol, 75-1398
Methoxychlor, 75-1341
Photodieldrin, 75-1340
Plants
Photodieldrin, 75-1340
SAN 6706, 75-0902
SAN 9789, 75-0902
Rat, 75-1376
Carbaryl, 75-1440
DDA, 75-1130
DDT, 75-1130
Ferbam, 75-1073
Hexachlorobenzene, 75-1360 75-1375
Hydroxyisoxazole, 75-1101
Methylmercuric chloride, 75-1132
Parathion, 75-1418
Phenothrin, 75-0928
Phenylmercuric chloride, 75-1132
Salicylate, 75-0947
2,4,5-T, 75-1361
Warfarin, 75-0947
Sharks/rays
DDT, 75-1350

Microsomes
In vitro
Pyrethrins, 75-1372
Rotenone, 75-1372

Mitochondria
see also Cytological effects
Animals/experimental
Chlordecone, 75-1342

Mitosis/meiosis
see also Cytological effects; Mutagenesis/teratogenesis
Human
Zineb, 75-1049
In vitro
Mercurials, 75-1115

Morbidity and mortality statistics
General
Paraquat, 75-0828
Belgium, 75-1331
Germany (DDR)
Arsenicals, 75-0813
Italy, 75-1312
Japan, 75-0809
Sweden
Amitrole, 75-0818, 75-0827
Tunisia
Parathion, 75-0830
United Kingdom

Morbidity and mortality statistics (cont'd)
Paraquat, 75-1067
USA-Iowa, 75-0898
USA-Louisiana
Hexachlorobenzene, 75-0762

Muscle, smooth
In vitro
Trichlorfon, 75-1332

Muscle, striated
Animals/experimental
Disulfoton, 75-1381
Methyl parathion, 75-0843
Organophosphates, 75-1082
In vitro
Trichlorfon, 75-1332

Musculoskeletal system
see Myocardium; Skeleton/bone

Mutagenesis/teratogenesis
see also Chromosomes/genes; Mitosis/meiosis
General, 75-0796, 75-1050
Carbamates, 75-1202
Carbaryl, 75-1202
Dalapon, 75-0964
Dichlorvos, 75-0832
MCPA, 75-1202
Animals/experimental
Captafol, 75-1174
Captan, 75-1174
Folpet, 75-1174
Trichlorfon, 75-1112
Human, 75-1407
In vitro
Aldrin, 75-1397
Phosphamidon, 75-1397
Microorganisms
Carbaryl, 75-0923, 75-1180 75-1396
Plants
Atrazine, 75-1355

Myocardium
see also Musculoskeletal system
Animals/experimental
Fenuron, 75-1171

Neonate
see also Reproduction/growth
Animals/experimental
Dichlorvos, 75-0824
Organochlorines, 75-1313
Parathion, 75-1080
TDE, 75-0925

Nervous system
see also Behavior; Brain; Demyelination; Peripheral nerves; Polyneuritis; Spinal cord
Animals/experimental
DDT, 75-1127
Dichlorvos, 75-1081
Dieldrin, 75-1391, 75-1403
Animals/non-target, 75-0898
Human
Organophosphates, 75-1048

Nucleic acids
see also Biochemical effects
Animals/experimental
Chlordane, 75-1074
Paraquat, 75-1173
Soman, 75-1091
In vitro
2,4-D, 75-1111
Mercurials, 75-1115
Microorganisms
Carbaryl, 75-1396
DDT, 75-1063

Peripheral nerves
see also Nervous system
Animals/experimental
Leptophos, 75-1096
Organophosphates, 75-1082

Photodecomposition
N-s-Butyl-4-*t*-butyl-2,6-dinitroaniline 75-1012
Chlordane, 75-0784
2,4-D, 75-0761
Dicamba, 75-0761
Dihydrochlordene dicarboxylic acid 75-1026
Diothyl, 75-0787
Methoprene, 75-1275
Methoprene, 75-1274
Nitrofen, 75-0777, 75-1273
Organochlorines, 75-1301
Organophosphates, 75-1282, 75-1301
Pyrethrins, 75-1016
Rotenone, 75-1301
Trifluralin, 75-1276

Placental transfer
see also Reproduction/growth
Animals/experimental
Amitrole, 75-1110
BHC isomers, 75-0943
Mercurials, 75-1324
Organochlorines, 75-1313
Polychlorinated biphenyls 75-0943
Human
Mercurials, 75-1324

Polyneuritis
see Nervous system

Porphyrins
see also Biochemical effects
General
Hexachlorobenzene, 75-0886
Polychlorinated biphenyls 75-0886
Animals/experimental
BHC, 75-0952
Hexachlorobenzene, 75-1131 75-1135, 75-1137, 75-1150 75-1151, 75-1375
Lindane, 75-1136
Polychloropinene, 75-1172
Human
Hexachlorobenzene, 75-1270

Prevention
General, 75-0810, 75-0814, 75-1239

Prevention (cont'd)
Disposal, 75-1304
Parathion, 75-0907
Education/training, 75-1047, 75-1235
Marten
Thallium, 75-0834
Protective equipment, 75-1047
Granosan, 75-1051
Safe packaging, 75-0811, 75-1304

Reproduction/growth
see also Eggshell effects; Embryo/-fetus; Estrogens; Fertility/sterility; Growth; Neonate; Placental transfer; Reproductive organs, female
Animals/experimental
DDT, 75-0848, 75-1085
Dieldrin, 75-0848, 75-0909
Metaldehyde, 75-1444
Organochlorines, 75-1183
Trichlorfon, 75-1419
Zineb, 75-1368
Ziram, 75-1368
Human
Organochlorines, 75-1313

Reproductive organs, female
see also Reproduction/growth
General, 75-1084
Animals/experimental
Paraquat, 75-0871

Reproductive organs, male, 75-1084
Animals/experimental
Dieldrin, 75-1133
Paraquat, 75-0871

Residue degradation
General
Organochlorines, 75-1293
Food and feed
Linuron, 75-0788
Plants
Phosalone, 75-1003
Soil
4-Aminopyridine, 75-1004
Atrazine, 75-1254
Carboxin, 75-0791
2,4-D, 75-1007
Ethion, 75-1003
Fensulfothion, 75-0765
Prometryne, 75-1254
Simazine, 75-1254
Water
Pyrazon, 75-1277

Residue dynamics, 75-0805, 75-0806
Dyfonate, 75-0820
Pyrazon, 75-1277

Residue removal
3cow
Organochlorines, 75-1023
Food and feed
Toxaphene, 75-1281
Plants
DDT, 75-0986
Endosulfan, 75-0986
TDE, 75-0986

Residue removal (cont'd)
Water, 75-0769
BHC, 75-1027
Captan, 75-1265
DDT, 75-1257
Herbicides, 75-1265
Organochlorines, 75-1265
Organophosphates, 75-1265
Propham, 75-1028
Simazine, 75-1027

Residues/air
General
DDT, 75-1250
Radosan, 75-1250
Industrial
Dimethoate, 75-1054
Methyl parathion, 75-1054
Trichlorfon, 75-1054
Rural, 75-0810
Trifluralin, 75-1276

Residues/food and feed
General, 75-0796, 75-1029, 75-1267
Arsenicals, 75-1284
BHC, 75-1268
Fenitrothion, 75-1248
Lead, 75-1284
Leptophos, 75-0782
Organochlorines, 75-1269
75-1284
Organophosphates, 75-1269
Phosmet, 75-1248
TCA, 75-0781
Total diet, 75-1033
Aldrin, 75-0785
BHC isomers, 75-0785
Chlordane, 75-0785
DDT, 75-0785
Dieldrin, 75-0785
Heptachlor, 75-0785
Heptachlor epoxide, 75-0785
Lindane, 75-0785
Organochlorines, 75-1292
Animal feed
BHC, 75-0773
DDT, 75-0773, 75-1030
Organochlorines, 75-1280
PCP, 75-1014
Cereals
Chlordane, 75-0759
Chlorpyrifos-methyl, 75-0771
2,4-D, 75-1260
Denmark, 75-0820
3,4-dichloroaniline, 75-0793
Dieldrin, 75-0776
Mercurials, 75-0776
Methyl bromide, 75-1013
Simazine, 75-0766
Dairy products, 75-1015
Aldrin, 75-1009
BHC, 75-1025, 75-1296
BHC isomers, 75-0760
Chlordane, 75-1009
Chlorpyrifos-methyl, 75-0771
DDT, 75-0760, 75-0763, 75-1005
75-1009, 75-1023, 75-1025
75-1296

Residues/food and feed (cont'd)
Dieldrin, 75-0760, 75-1005
75-1009
Endosulfan, 75-1009
Heptachlor, 75-1009
Heptachlor epoxide, 75-0760
75-1009
Lindane, 75-1023
Organochlorines, 75-1298
Polychlorinated biphenyls
75-1005
Fruits, 75-1015, 75-1017
Arsenicals, 75-0763, 75-1286
Azinphosethyl, 75-1255
Azinphosmethyl, 75-1255
Captan, 75-1294
Ethylene dibromide, 75-0783
Fenitrothion, 75-1286
Lead, 75-1286
Methoxychlor, 75-1294
Organochlorines, 75-1298
Meat
DDT, 75-0770
Dieldrin, 75-0770
Hexachlorobenzene, 75-1271
Lead, 75-0770
Mercurials, 75-0770
Organochlorines, 75-1263
75-1272, 75-1298
Poultry
DDT, 75-1261
Hexachlorobenzene, 75-1271
PCP derived compounds, 75-0872
Vegetables, 75-1015
Carbamates, 75-0804
Chlordane, 75-0759
Chlorpropham, 75-0764
Chlorpyrifos, 75-1040
Denmark, 75-0820
Fensulfothion, 75-0765
Fungicides, 75-0804
Leptophos, 75-1040
Organochlorines, 75-1298
Propham, 75-0764
Pyrazon, 75-1285
Simazine, 75-0766

Residues/humans
General
Hexachlorobenzene, 75-0762
Organochlorines, 75-1037
Adipose, 75-1264, 75-1289
DDE, 75-1262
DDT, 75-1262, 75-1289
Organochlorines, 75-0763
75-1290, 75-1297
Blood
DDT, 75-1022, 75-1258
Hexachlorobenzene, 75-1270
Organochlorines, 75-1283
75-1313
Polychlorinated biphenyls
75-1258
Milk
BHC isomers, 75-0760
DDE, 75-1288
DDT, 75-0760, 75-1288, 75-1289
Dieldrin, 75-0760

Residues/humans (cont'd)
Heptachlor epoxide, 75-0760
Organochlorines, 75-1284
75-1297
Urine
Demeton, 75-1329
Mercurials, 75-1329
Parathion, 75-1329

Residues/non-target organisms
General
Aldrin, 75-0799
BHC isomers, 75-0799
2,4-D, 75-0798
DDE, 75-0799
Dieldrin, 75-0799
Heptachlor, 75-0799
Heptachlor epoxide, 75-0799
Mirex, 75-1034, 75-1035
2,4,5-T, 75-0798
TFM, 75-0906
Amphibians
DDT, 75-1259
Birds
DDT, 75-1259
Mercurials, 75-0774, 75-1031
Organochlorines, 75-0772
75-0774, 75-1010, 75-1297
75-1299
Chicken
PCP derived compounds, 75-0779
Crustacea
DDE, 75-1279
DDT, 75-1008, 75-1279
Organochlorines, 75-0786
75-1249
Polychlorinated biphenyls
75-0786, 75-1008
Eggs
Mercurials, 75-0774
Organochlorines, 75-0774
75-1297, 75-1300
PCP derived compounds, 75-0779
Fish
DDT, 75-1008, 75-1032, 75-1251
Dieldrin, 75-1251
Organochlorines, 75-0786
75-1010, 75-1249, 75-1299
Polychlorinated biphenyls
75-0786, 75-1008, 75-1032
Molluscs
Organochlorines, 75-1010
75-1249
Pig
Organochlorines, 75-1298
Plankton/algae
Polychlorinated biphenyls
75-0775
Rabbit
Agallol, 75-0800
Reptiles
DDT, 75-1259
Organochlorines, 75-0772
Shrew
Organochlorines, 75-0772
Wolf
Organochlorines, 75-1038

Residues/plants
General, 75-1029
2,4-D, 75-0798
Mirex, 75-1036
2,4,5-T, 75-0798
Forest
Granulosis virus, 75-0797
Medicinals and condiments
DDT, 75-0803
Herbicides, 75-0803
Organophosphates, 75-0803
Tobacco
Acephate, 75-1278
Carbaryl, 75-0780
Carbofuran, 75-0780
Leptophos, 75-0780
Organochlorines, 75-0780

Residues/soil
General, 75-1029
Agallol, 75-0800
Atrazine, 75-1254
2,4-D, 75-1260
DDT, 75-1030
Denmark, 75-0820
Dichlobenil, 75-0795
Diuron, 75-0795
Herbicides, 75-0789
Mercurials, 75-1019
Mirex, 75-1036
Picloram, 75-1039
Prometryne, 75-1251, 75-1254
Propanil, 75-0793
Simazine, 75-0766, 75-0795
75-1254
Terbacil, 75-0795
Terbutryne, 75-0794
Adsorption
DDT, 75-1253
Diquat, 75-0802
Fenuron, 75-1024
Linuron, 75-1006
Monuron, 75-1024
Paraquat, 75-0802, 75-0807
Picloram, 75-0767, 75-1011
Movement
Fungicides, 75-1021
Herbicides, 75-0790
Organochlorines, 75-1295
Prometryne, 75-1287
Volatilization
Trifluralin, 75-0778

Residues/water
General
2,4-D, 75-0798
Organochlorines, 75-0801
75-1291, 75-1299
2,4,5-T, 75-0798
Groundwater/rain
DDT, 75-1266
Lakes/ponds, 75-1274
Mirex, 75-1036
Oceans/seas
DDT, 75-1252, 75-1253
Organochlorines, 75-0786
Polychlorinated biphenyls
75-0786

Residues/water (cont'd)
Rivers/streams
BHC, 75-1018
Dicamba, 75-1256
TCA, 75-1018

Respiration, cellular
see also Biochemical effects
Amino acids/peptides/proteins
Dichlorvos, 75-1093
Animals/experimental
DDT, 75-0869, 75-0870
Malathion, 75-1424
Parathion, 75-1109
Human
Copper sulfate, 75-1042
In vitro
Fluorine compounds, 75-1186
Mercurials, 75-1186
Organophosphates, 75-1186
Parathion, 75-1092
PCP, 75-0861
TFM, 75-1351
Microorganisms
Chlorpyrifos, 75-1203
Coumaphos, 75-1203
Dichlorvos, 75-1203
Dowco 214, 75-1203
Dowco 217, 75-1203
Fensulfothion, 75-1203
Malathion, 75-1203
Naled, 75-1203
Phenamiphos, 75-1203

Respiratory system
see also Gills; Lung; Upper respiratory tract
Animals/experimental
Fenuron, 75-1171
Plictran, 75-0949
Human
Arsenicals, 75-1303

Reticuloendothelial system
see also Marrow
Animals/experimental
Dimethoate, 75-0841
Trichlorfon, 75-0841

Reviews
General, 75-1243
Arsenicals, 75-1001
Mercurials, 75-1001
Analysis
Arsenicals, 75-1001
Herbicides, 75-0967
Mercurials, 75-1001
Epidemiology, prevention, and treatment, 75-1307
Organophosphates, 75-0817
Monitoring and residues, 75-0805
Carbamates, 75-0804
Fungicides, 75-0804
Toxicology and pharmacology
75-0806, 75-0883, 75-1100
75-1168, 75-1196, 75-1336
75-1429, 75-1430, 75-1431
75-1432, 75-1433, 75-1434
Dalapon, 75-0964

Reviews (cont'd)
Hexachlorobenzene, 75-1137
Mercurials, 75-1358
Organophosphates, 75-0959 75-1157

Safety standards
General, 75-1050
Mercurials, 75-1000
Methyl bromide, 75-1311
Acceptable daily intake, 75-0796 75-1247
Reentry time, 75-0826, 75-1236
TLV/MAC, 75-0796, 75-1053
Cuprozin, 75-1144
Milbex, 75-1143
Toxaphene, 75-1281
Tolerances, 75-0826, 75-0838 75-1053
Toxicity rating class
Paraquat, 75-1314

Sensory system
see Vision

Shrew
Residues/non-target organisms
Organochlorines, 75-0772

Skeleton/bone
see Musculoskeletal system

Skin
see also Integument
Animals/experimental
Captafol, 75-1189
Cuprozin, 75-1144
Dichlozoline, 75-1145
Zineb, 75-1189
Human
Dioxins, 75-1306

Spectrometry
see also Analysis
Colorimetry
Biphenyl, 75-1449
Carbamates, 75-1214, 75-1494
Carbaryl, 75-1481
DDE, 75-1461
DDT, 75-1461
Dichlorvos, 75-0966, 75-1491 75-1493
Herbicides, 75-1212
Malathion, 75-0966
MCPA, 75-1459
Organochlorines, 75-1477
Organophosphates, 75-1494
Polychlorinated biphenyls 75-1477
Pyrethrins, 75-0977
Ronnel, 75-0966
Rotenone, 75-0977
TDE, 75-1461
Tetrachlorvinphos, 75-0966
Trichlorfon, 75-1491
Fluorometry
Coumaphos, 75-0975
1-Naphthaleneacetic acid, 75-1218
Quinalphos, 75-0975
Triazines, 75-1479

Spectrometry (cont'd)
Infrared
Alachlor, 75-1457
Chlorpropham, 75-0995
Propham, 75-0995
Mass spectrometry
Organophosphates, 75-0994
NMR
Pyrethrins, 75-0965
Radiometry
Aldrin, 75-1487
Atrazine, 75-0991
Carbamates, 75-0969
Chloramben, 75-0991
Dicamba, 75-0991
Dieldrin, 75-1487
Organochlorines, 75-1229
Organophosphates, 75-0969
UV
Benomyl, 75-0988
2,4-D, 75-1465
DDT, 75-1452
Dimethoate, 75-1465
Fluometuron, 75-1484
Trichlorfon, 75-1465

Spinal cord
see also Nervous system
Animals/experimental
DDT, 75-0851
Leptophos, 75-1096

Spleen
Animals/experimental
Fenuron, 75-1171
2,4,5-T, 75-0904

Therapeutic use
Cancer
DDT, 75-1197
Picloram, 75-1334
TDE, 75-0874, 75-1335
Cushing's syndrome
DDT, 75-1197
TDE, 75-1160
Scabies
BHC, 75-1121

Thyroid
Animals/experimental
Amitrole, 75-1410
Fenuron, 75-1171

Titration
see also Analysis
Dimethirimol, 75-1453
Organochlorines, 75-1482
Pirimicarb, 75-1453

Toxicity/experimental animals
General
2,4-D, 75-0938
Dalapon, 75-0964
DDT derived compounds 75-0910
Endosulfan, 75-1389
Methoxychlor derived compounds 75-0910
Organochlorines, 75-1301
Organophosphates, 75-0961

Toxicity/experimental animals (cont'd)
Organophosphates, 75-0963 75-1301
Propoxur, 75-1065
Pyrethrins, 75-1016
Rotenone, 75-1301
TFM, 75-0906
Amphibians
Malathion, 75-1416
Trichlorfon, 75-1416
Birds
4-Aminopyridine, 75-0918
Chlordecone, 75-1439
Cow, 75-0898
Crustacea
Abate, 75-0868
Carbaryl, 75-1382
DDT, 75-1166
Diquat, 75-1357
Dog
4-Aminopyridine, 75-0918
Antimycin, 75-0901
Fish
Abate, 75-0922
Akton, 75-0922
Aldrin, 75-1095
Atrazine, 75-0911
Bay-69047, 75-0922
Carbaryl, 75-0911, 75-1382
Chlorpyrifos, 75-0922
2,4-D, 75-0911, 75-1383
DDT, 75-0911
Dichlorvos, 75-0842
Dieldrin, 75-0911
Endosulfan, 75-0911
Fenitrothion, 75-1163, 75-1164
Herbicides, 75-1356
Hymexazol, 75-1398
Malathion, 75-0911
Mirex, 75-1209
Naled, 75-0950
Organochlorines, 75-1183 75-1345
Organophosphates, 75-1345
Phoxim, 75-0922
TFM, 75-1351
Guinea pig
Dieldrin, 75-1391, 75-1403
Insects
Fluenethyl, 75-1353
Microorganisms
2,4-D, 75-0937
Dalapon, 75-1154
D-D, 75-0873
DDE, 75-0864
DDT, 75-0864, 75-0937
Ethoprop, 75-0873
Malathion, 75-1413
Methoxychlor, 75-0937
Methyl demeton, 75-0937
3-Methoxy-4,6-bis(isopropylamino-)-*S*-triazi, 75-0937
Organochlorines, 75-1182
Paraquat, 75-1154
PCP, 75-0937
Polychlorinated biphenyls 75-0864, 75-1182

Toxicity/experimental animals (cont'd)
2,4,5-T, 75-0937
Thiram, 75-1156
Trichlorfon, 75-0937, 75-1413
Molluscs
Carbaryl, 75-1382
Monkey, 75-1178
Mouse
Dioxins, 75-1437
Fluenethyl, 75-1353
Heptachlor, 75-1346
Hymexazol, 75-1398
NS 2662, 75-1400
Promecarb, 75-1365
Toxaphene, 75-1281
Pheasant, 75-1387
Organophosphates, 75-1387
Pig
Dichlorvos, 75-0824
Plankton/algae
DDT, 75-0908
Polychlorinated biphenyls 75-0908
Plants
Ametryne, 75-0897
Diuron, 75-0897
Fluometuron, 75-0897
Prometryne, 75-0897
Terbutryne, 75-0794
Rabbit
MCPP, 75-1443
Rat
Acephate derived compounds 75-0957
4-Aminopyridine, 75-0918
Antimycin, 75-0901
Dimethoate, 75-1380
DNBP, 75-1369
Formetanate, 75-1366
Hymexazol, 75-1398
Lindane, 75-1369
Malathion, 75-1380
MCPA, 75-1443
Metaldehyde, 75-1444
Molluscicides, 75-1441
Parathion, 75-1418
Promecarb, 75-1365
Propoxur, 75-1348
Thiometon, 75-1364
Toxaphene, 75-1281
Sharks/rays
DDT, 75-1350
Sheep
Plictran, 75-0949

Toxicity/humans
General, 75-1310, 75-1312
Dichlorvos, 75-0832
Paraquat, 75-0831, 75-1045 75-1314
Thallium, 75-1060
Accidental, 75-0811, 75-0837
Arsenicals, 75-1043
Carbamates, 75-1304
Chlordane, 75-1323
Chlorfenvinphos, 75-1326
Herbicides, 75-0792
Mercurials, 75-0836, 75-1324

Toxicity/humans (cont'd)
Mercurials, 75-1328
Organophosphates, 75-1304 75-1305
Oxamyl, 75-1304
Paraquat, 75-1059, 75-1319
Parathion, 75-1304
Phosphorus, 75-0821
Toxaphene, 75-0839
Intentional
Calcium polysulfide, 75-1322
Copper sulfate, 75-1042
Diazinon, 75-1056
Mevinphos, 75-1044
Organophosphates, 75-1046
Paraquat, 75-0829, 75-1059 75-1318
Phosphorus, 75-0821
Occupational, 75-0808, 75-0835 75-1050, 75-1057, 75-1058 75-1307, 75-1445
Amitrole, 75-0818, 75-0827
Arsenicals, 75-0816, 75-1303
Azinphosmethyl, 75-0823
Bordeaux mixture, 75-1041
Carbamates, 75-0816, 75-1321
Carbaryl, 75-1047
Chlordane, 75-0815, 75-1047
Copper, 75-1052
Copper sulfate, 75-1041
2,4-D, 75-1325
DDT, 75-0812, 75-0815
Demeton, 75-1329
Diazinon, 75-1047
Dichlorvos, 75-1327
Dimethoate, 75-1054
Dioxins, 75-1306
Ethion, 75-0823
Ethylene oxide, 75-1395
Formothion, 75-1052
Granosan, 75-1052
Lead, 75-0816
Lindane, 75-0812, 75-0815 75-1055
MCPA, 75-1302, 75-1325
Mercurials, 75-0816, 75-1329
Methyl bromide, 75-1311
Methyl parathion, 75-1054
Naled, 75-0823
Organochlorines, 75-0816 75-1315, 75-1316
Organophosphates, 75-0812 75-0816, 75-0822, 75-1046 75-1315, 75-1316, 75-1321
Paraquat, 75-0828
Parathion, 75-1329
PCP, 75-1107
Phosalone, 75-0823
Propham, 75-1327
Thiometon, 75-1364
Thiram, 75-1320
Trichlorfon, 75-1054
Zineb, 75-1049

Toxicity/non-target organisms
General, 75-0835, 75-1331
Herbicides, 75-0792
Strychnine, 75-1309

Toxicity/non-target organisms (cont'd)
Badger
Thallium, 75-0834
Birds
Dieldrin, 75-1113
Organochlorines, 75-0768
Cow
Arsenicals, 75-1330
Mercurials, 75-0840
Toxaphene, 75-1330
Eggs
Organochlorines, 75-0768
Fox
Thallium, 75-0833

Treatment of poisoning
General
Lindane, 75-1055
MCPA, 75-1302
Paraquat, 75-0829, 75-0831
Phosphorus, 75-0821
Adsorbents
Paraquat, 75-1317
Atropine
Organophosphates, 75-1048
Bentonite and purgatives
Paraquat, 75-0862
α-Lipoic acid
Mercurials, 75-1427
Oximes
Chlorfenvinphos, 75-1326
Organophosphates, 75-0817
Prussian blue
Thallium, 75-1060
Superoxide dismutase and *d*-propranolol
Paraquat, 75-1067

Upper respiratory tract
see Respiratory system

Vision
see also Sensory system
Animals/experimental
Captafol, 75-1189
Chlorfenvinphos, 75-1399
DFP, 75-1333
Disulfoton, 75-1338, 75-1381
Fenitrothion, 75-0942, 75-1190
Organophosphates, 75-1188
Zineb, 75-1189
Human
Organophosphates, 75-1048 75-1305
Sarin, 75-1139

Vitamins/coenzymes
see Biochemical effects

Subject Index: Compounds

Abate
Toxicity/experimental animals
Crustacea, 75-0868
Fish, 75-0922

Acephate
Residues/plants
Tobacco, 75-1278

Acephate derived compounds
Toxicity/experimental animals
Rat, 75-0957

Acrolein
see also Herbicides
Blood pressure
Animals/experimental, 75-0945

Agallol
see also Herbicides
Residues/non-target organisms
Rabbit, 75-0800
Residues/soil
General, 75-0800

Akton
Toxicity/experimental animals
Fish, 75-0922

Alachlor
see also Herbicides
Biotransformation
Fungi, 75-0893
Spectrometry
Infrared, 75-1457

Alavit 55
see DDT

Aldrin
see also Organochlorines
Chromosomes/genes
In vitro, 75-1397
Enzyme activity
Mixed function oxidases, 75-1095
Factors influencing metabolism/toxicity
Age, 75-1072
Growth
Microorganisms, 75-1394
Metabolism
In vitro, 75-1072
Mutagenesis/teratogenesis
In vitro, 75-1397
Residues/food and feed
Total diet, 75-0785
Dairy products, 75-1009
Residues/non-target organisms
General, 75-0799
Spectrometry
Radiometry, 75-1487
Toxicity/experimental animals
Fish, 75-1095

Alkanes
Biotransformation
Microorganisms, 75-0846

Altosid
see Methoprene

Ametryne
see also Triazines
Toxicity/experimental animals
Plants, 75-0897

4-Aminopyridine
see also Repellents
Residue degradation
Soil, 75-1004
Toxicity/experimental animals
Birds, 75-0918
Dog, 75-0918
Rat, 75-0918

Amitrole
see also Herbicides
Carcinogenesis
General, 75-0818, 75-0827
Animals/experimental, 75-1409 75-1410
Distribution/storage
Mouse, 75-1110
Embryo/fetus
Animals/experimental, 75-1110
Enzyme activity
Catalase, 75-0900, 75-0946
Factors influencing metabolism/toxicity
Interactions, 75-0946, 75-1409
Thyroidectomy, 75-1410
Liver
Animals/experimental, 75-1409 75-1410
Lung
Human, 75-0818
Morbidity and mortality statistics
Sweden, 75-0818, 75-0827
Placental transfer
Animals/experimental, 75-1110
Thyroid
Animals/experimental, 75-1410
Toxicity/humans
Occupational, 75-0818, 75-0827

Aniline
see also Herbicides
Metabolism
Microorganisms, 75-1359

Antibiotics
Bioassay, 75-1458

Antidotes
see Pralidoxime

Antimycin
Toxicity/experimental animals
Dog, 75-0901
Rat, 75-0901

Armine
Acid-base balance
Animals/experimental, 75-1446
Enzyme activity
Cholinesterase, 75-1446

Arsenicals
see also Cacodylic acid; Lead arsenate; Sodium arsenite
Blood cells
Human, 75-1043
Carcinogenesis
Human, 75-0813, 75-1303
Environmental pollution, 75-1001
Growth
Microorganisms, 75-0954
Morbidity and mortality statistics
Germany (DDR), 75-0813
Residues/food and feed
General, 75-0763 75-1284
Fruits, 75-1286
Respiratory system
Human, 75-1303
Reviews
General, 75-1001
Analysis, 75-1001
Toxicity/humans
Accidental, 75-1043
Occupational, 75-0816, 75-1303
Toxicity/non-target organisms
Cow, 75-1330

Atratone
see Triazines

Atrazine
see also Herbicides; Triazines
Chromatography
Gas-liquid, 75-0976
Thin-layer, 75-1216
Metabolism
Plankton/algae, 75-1341
Mutagenesis/teratogenesis
Plants, 75-1355
Residue degradation
Soil, 75-1254
Residues/soil
General, 75-1254
Spectrometry
Radiometry, 75-0991
Toxicity/experimental animals
Fish, 75-0911

Atropine
Blood cells
Animals/experimental, 75-1390
Enzyme activity
General, 75-1390
Heart
Animals/non-target, 75-1390

Avicides
see 2-Chloro-4-acetotoluidine;3-chloro-*p*-toluidine

Azinphosethyl
see also Insecticides
Residues/food and feed
Fruits, 75-1255

Azinphosmethyl
Chromosomes/genes
In vitro, 75-0881

Azinphosmethyl (cont'd)
Enzyme activity
Cholinesterase, 75-0823, 75-0863
Factors influencing metabolism/toxicity
Taxon, 75-0863
Residues/food and feed
Fruits, 75-1255
Toxicity/humans
Occupational, 75-0823

Bay-69047
Toxicity/experimental animals
Fish, 75-0922

Bay 94337
see Metribuzin
Chromatography
Gas-liquid, 75-0985

Baygon
see Propoxur

Benomyl
see also Fungicides
Bioassay, 75-0988
Biochemical effects
Microorganisms, 75-1204
Biotransformation
Annelids, 75-0929
Microorganisms, 75-1204
Chromatography
Thin-layer, 75-0996
Enzyme activity
Cholinesterase, 75-0929
Dehydrogenase, 75-1204
Spectrometry
UV, 75-0988

Benthiocarb
see also Herbicides
Biotransformation
In vitro, 75-1192
Mouse, 75-1192

BHC
see also Organochlorines
Biochemical effects
Animals/experimental, 75-1169
Carcinogenesis
Animals/experimental, 75-1411 75-1412
Chromatography
Thin-layer, 75-1489
Enzyme activity
General, 75-1170
Mixed function oxidases, 75-1169
Factors influencing metabolism/toxicity
Interactions, 75-1411, 75-1412
Sex, 75-1412
Taxon, 75-1412
Growth
Microorganisms, 75-1394
Liver
Animals/experimental, 75-1169 75-1411, 75-1412
Porphyrins
Animals/experimental, 75-0952
Residue removal
Water, 75-1027
Residues/food and feed
General, 75-1268
Animal feed, 75-0773
Dairy products, 75-1025, 75-1296
Residues/water
Rivers/streams, 75-1018
Therapeutic use
Scabies, 75-1121

γ-BHC
see Lindane

BHC isomers
see also Organochlorines
Enzyme activity
Mixed function oxidases, 75-0924
Excretion
Mouse, 75-0943
Factors influencing metabolism/toxicity
Taxon, 75-0921
Placental transfer
Animals/experimental, 75-0943
Residues/food and feed
Total diet, 75-0785
Dairy products, 75-0760
Residues/humans
Milk, 75-0760
Residues/non-target organisms
General, 75-0799

Biphenyl
Biotransformation
In vitro, 75-1352
Spectrometry
Colorimetry, 75-1449

Birlane
see Chlorfenvinphos

Bombykol
Biotransformation
Insects, 75-0914
Chromatography
Thin-layer, 75-0914

Bordeaux mixture
see also Fungicides
Digestive system
Animals/experimental, 75-0919
Lung
Human, 75-1041
Toxicity/humans
Occupational, 75-1041

Botanicals
see Dimethrin; Rotenone

Bromacil
Biochemical effects
Microorganisms, 75-0934

Bromophos
Biotransformation
In vitro, 75-1423
Factors influencing metabolism/toxicity
Taxon, 75-1423

***N*-*s*-Butyl-4-*t*-butyl-2,6-dinitroaniline**
Photodecomposition, 75-1012

Butylate
see also Herbicides
Biotransformation
In vitro, 75-1192
Mouse, 75-1192

Cacodylic acid
see Arsenicals

Calcium polysulfide
Toxicity/humans
Intentional, 75-1322

Captafol
see also Fungicides
Biochemical effects
Animals/experimental, 75-1189
Growth
Animals/experimental, 75-1189
Mutagenesis/teratogenesis
Animals/experimental, 75-1174
Skin
Animals/experimental, 75-1189
Vision
Animals/experimental, 75-1189

Captan
see also Fungicides
Enzyme activity
Acid phosphatase, 75-1414
Factors influencing metabolism/toxicity
Nutritional state, 75-1414
Mutagenesis/teratogenesis
Animals/experimental, 75-1174
Residue removal
Water, 75-1265
Residues/food and feed
Fruits, 75-1294

Carbamates
see also Carbaryl; Carbofuran; Karbutilate; Maneb; Methiocarb; Promecarb; Zineb; Ziram
Amino acids/peptides/proteins
Animals/experimental, 75-0850
Analysis
General, 75-0968
Biotransformation
Insects, 75-1434
Chromatography
Column, 75-1222
Gas-liquid, 75-1213, 75-1220 75-1469
Enzyme activity
Aliesterase, 75-1434
Cholinesterase, 75-0858, 75-0968 75-1321
Esterases, 75-1118
Mixed function oxidases, 75-1434
Enzyme assay
Cholinesterase, 75-1494
Experimental design
Toxicology and pharmacology 75-0858
Factors influencing metabolism/toxicity

Carbamates (cont'd)
Adaptation, 75-1434, 75-1436
Molecular structure, 75-1118
Sex, 75-1162
Growth
Animals/experimental, 75-0850
Liver
Animals/experimental, 75-0850
Metabolism
Insects, 75-1436
Mutagenesis/teratogenesis
General, 75-1202
Residues/food and feed
Vegetables, 75-0804
Reviews
Monitoring and residues, 75-0804
Spectrometry
Colorimetry, 75-1214, 75-1494
Radiometry, 75-0969
Toxicity/humans
Accidental, 75-1304
Occupational, 75-0816, 75-1321

Carbaryl
see also Carbamates
Absorption
Rat, 75-0844
Analysis
Sample preparation, 75-1481
Biotransformation
Fungi, 75-1140
Microorganisms, 75-1141
Rat, 75-1423
Chromosomes/genes
Microorganisms, 75-1396
Digestive system
Animals/experimental, 75-0919
Distribution/storage
Rat, 75-0780
Enzyme activity
General, 75-0877
Cholinesterase, 75-1162
Experimental design
Toxicology and pharmacology 75-0844
Factors influencing metabolism/toxicity
Age, 75-1377
Interactions, 75-1140
Sex, 75-1064, 75-1377
Metabolism
Human, 75-1373
In vitro, 75-1373
Insects, 75-1377
Microorganisms, 75-1354
Plankton/algae, 75-1341
Rat, 75-1440
Mutagenesis/teratogenesis
General, 75-1202
Microorganisms, 75-0923, 75-1180 75-1396
Nucleic acids
Microorganisms, 75-1396
Residues/plants
Tobacco, 75-0780
Spectrometry
Colorimetry, 75-1481
Toxicity/experimental animals

Carbaryl (cont'd)
Crustacea, 75-1382
Fish, 75-0911, 75-1382
Molluscs, 75-1382
Toxicity/humans
Occupational, 75-1047

Carbetox
see Malathion

Carbofuran
see also Carbamates
Chromatography
Gas-liquid, 75-1467
Distribution/storage
Rat, 75-0780
Enzyme activity
Cholinesterase, 75-1104
Residues/plants
Tobacco, 75-0780

Carbon tetrachloride
see Fumigants

Carboxin
see also Fungicides
Distribution/storage
Plants, 75-0791
Residue degradation
Soil, 75-0791

Chemosterilants
see Tepa

Chloramben
see also Herbicides
Spectrometry
Radiometry, 75-0991

Chloramphenicol
Liver
Human, 75-1308

Chlordane
see also Organochlorines
Amino acids/peptides/proteins
Human, 75-0815
Cytological effects
Human, 75-1323
Lipids/steroids/sterols
Human, 75-0815
Liver
Animals/experimental, 75-1074
Human, 75-1323
Nucleic acids
Animals/experimental, 75-1074
Photodecomposition, 75-0784
Residues/food and feed
Total diet, 75-0785
Cereals, 75-0759
Dairy products, 75-1009
Vegetables, 75-0759
Toxicity/humans
Accidental, 75-1323
Occupational, 75-0815, 75-1047

Chlordecone
see also Insecticides
Enzyme activity
ATPase, 75-1342
Factors influencing metabolism/toxicity

Chlordecone (cont'd)
Age, 75-1439
Light/radiation, 75-1439
Mitochondria
Animals/experimental, 75-1342
Toxicity/experimental animals
Birds, 75-1439

Chlordimeform
Metabolism
In vitro, 75-1374

Chlorfenvinphos
see also Organophosphates
Brain
Animals/experimental, 75-1399
Enzyme activity
Cholinesterase, 75-1399
Toxicity/humans
Accidental, 75-1326
Treatment of poisoning
Oximes, 75-1326
Vision
Animals/experimental, 75-1399

2-Chloro-4-acetotoluidine
see also Avicides
Biotransformation
Rat, 75-1078
Factors influencing metabolism/toxicity
Taxon, 75-1078

3-Chloro-*p*-toluidine
see also Avicides
Biotransformation
Chicken, 75-1078
Factors influencing metabolism/toxicity
Taxon, 75-1078

5-*p*-Chlorophenyl silatrane
Factors influencing metabolism/toxicity
Sex, 75-1167

Chlorpropham
see also Herbicides
Residues/food and feed
Vegetables, 75-0764
Spectrometry
Infrared, 75-0995

Chlorpyrifos
Residues/food and feed
Vegetables, 75-1040
Respiration, cellular
Microorganisms, 75-1203
Toxicity/experimental animals
Fish, 75-0922

Chlorpyrifos-methyl
Enzyme activity
Cholinesterase, 75-0771
Excretion
Cow, 75-0771
Growth
Animals/experimental, 75-0771
Residues/food and feed
Cereals, 75-0771
Dairy products, 75-0771

Chlortoluron
Enzyme activity
General, 75-1422, 75-1425
Liver
Animals/experimental, 75-1422
75-1425

Cisanilide
see also Herbicides
Biotransformation
In vitro, 75-1195

Copper
see also Heavy metals
Enzyme activity
Cholinesterase, 75-1142
Factors influencing metabolism/
toxicity
Interactions, 75-1052, 75-1142
Kidney
Animals/experimental, 75-1142
Toxicity/humans
Occupational, 75-1052

Copper oxychloride
Enzyme activity
Cytochrome oxidase, 75-1404

Copper sulfate
Lung
Human, 75-1041
Respiration, cellular
Human, 75-1042
Toxicity/humans
Intentional, 75-1042
Occupational, 75-1041

Cotoran
see Fluometuron

Coumaphos
see also Insecticides
Enzyme activity
Cholinesterase, 75-0915
Growth
Animals/experimental, 75-0915
Respiration, cellular
Microorganisms, 75-1203
Spectrometry
Fluorometry, 75-0975

Crimidine
Chromatography
Thin-layer, 75-1219

Crufomate
Amino acids/peptides/proteins
Animals/experimental, 75-0850
Growth
Animals/experimental, 75-0850
Liver
Animals/experimental, 75-0850

Cruformate
see Organophosphates

Cuprozin
Embryo/fetus
Animals/experimental, 75-1144
Enzyme activity
General, 75-1144
Safety standards

Cuprozin (cont'd)
TLV/MAC, 75-1144
Skin
Animals/experimental, 75-1144

Cyanazine
see also Herbicides
Biotransformation
Plants, 75-0903

Cycloate
Biotransformation
In vitro, 75-1192
Mouse, 75-1192

2,4-D
Biotransformation
Microorganisms, 75-1124
Mouse, 75-1105
Rat, 75-1105
Chromatography
Thin-layer, 75-1460
EEG
Human, 75-1325
Factors influencing metabolism/
toxicity
Formulation, 75-1383
Lipids/steroids/sterols
In vitro, 75-0854
Metabolism
Plankton/algae, 75-1341
Nucleic acids
In vitro, 75-1111
Photodecomposition, 75-0761
Residue degradation
Soil, 75-1007
Residues/food and feed
Cereals, 75-1260
Residues/non-target organisms
General, 75-0798
Residues/plants
General, 75-0798
Residues/soil
General, 75-1260
Residues/water
General, 75-0798
Spectrometry
UV, 75-1465
Toxicity/experimental animals
General, 75-0938
Fish, 75-0911, 75-1383
Microorganisms, 75-0937
Toxicity/humans
Occupational, 75-1325

Dalapon
see also Herbicides
Chromatography
Thin-layer, 75-1460
Enzyme activity
Urease, 75-1154
Growth
Microorganisms, 75-1154
Metabolism
General, 75-0964
Mutagenesis/teratogenesis
General, 75-0964

Dalapon (cont'd)
Reviews
Toxicology and pharmacology
75-0964
Toxicity/experimental animals
General, 75-0964
Microorganisms, 75-1154

DBCP
Growth
Microorganisms, 75-1379

DCPA
see also Herbicides
Amino acids/peptides/proteins
Animals/experimental, 75-0892
Carbohydrates
Animals/experimental, 75-0892
Electrolytes
Animals/experimental, 75-0892

D-D
see also Fumigants
Growth
Microorganisms, 75-1379
Toxicity/experimental animals
Microorganisms, 75-0873

DDA
Metabolism
Rat, 75-1130

DDD
see TDE

DDE
see also Organochlorines
Analysis
Sample preparation, 75-0980
Eggshell effects
Animals/experimental, 75-0849
75-1097, 75-1148
Electrolytes
Animals/experimental, 75-1066
75-1097
Enzyme activity
ATPase, 75-1066, 75-1097
75-1148
Carbonic anhydrase, 75-1097
75-1148
Excretion
Duck, 75-0865
Factors influencing metabolism/
toxicity
Interactions, 75-0864
Glands
Animals/non-target, 75-0865
Residues/humans
Adipose, 75-1262
Milk, 75-1288
Residues/non-target organisms
General, 75-0799
Crustacea, 75-1279
Spectrometry
Colorimetry, 75-1461
Toxicity/experimental animals
Microorganisms, 75-0864

DDT
see also Organochlorines
Absorption

DDT (cont'd)
Crustacea, 75-1147
Fish, 75-0916, 75-1146
Amino acids/peptides/proteins
Human, 75-0815
In vitro, 75-1193
Analysis
Sample preparation, 75-0980
Balance
Animals/experimental, 75-1165
Behavior
Animals/experimental, 75-1147 75-1165, 75-1166, 75-1208 75-1210
Biotransformation
Insects, 75-1434
Microorganisms, 75-1141
Brain
Animals/experimental, 75-0932
Carbohydrates
Animals/experimental, 75-1337
In vitro, 75-0853
Carcinogenesis
Animals/experimental, 75-0867 75-1411
Human, 75-0812
Cell membranes
Microorganisms, 75-1063
Chromatography
Gas-liquid, 75-1454
Thin-layer, 75-0981, 75-1216 75-1452, 75-1489
Cytochromes
Animals/experimental, 75-1433
Digestive system
Animals/experimental, 75-0919
Distribution/storage
Birds, 75-0866
Crustacea, 75-1438
Duck, 75-1385
Fish, 75-1146
Mouse, 75-1401, 75-1402
Rat, 75-0780, 75-1161
Electrolytes
Animals/experimental, 75-1126
Embryo/fetus
Animals/experimental, 75-1146
Human, 75-1022
Endoplasmic reticulum
Animals/experimental, 75-0887 75-1371
Enzyme activity
General, 75-1371, 75-1420
Aliesterase, 75-1434
ATPase, 75-0845, 75-1122
Cholinesterase, 75-0851
Mixed function oxidases, 75-0848 75-0884, 75-1198, 75-1201 75-1337, 75-1433, 75-1434
Ornithine decarboxylase, 75-1094
Excretion
Chicken, 75-1125
Duck, 75-0865
Rat, 75-1161
Experimental design
Monitoring and residues, 75-1252
Factors influencing metabolism/ toxicity
Adaptation, 75-1434, 75-1436
Disease state, 75-1385
Interactions, 75-0864, 75-1198 75-1411
Nutritional state, 75-1127
Taxon, 75-1125, 75-1193, 75-1210
Temperature, 75-0916
Glands
Animals/non-target, 75-0865
Growth
Animals/experimental, 75-0850 75-0884
Microorganisms, 75-1063, 75-1394
Immunology
Animals/experimental, 75-1385
Kidney
In vitro, 75-0853
Laws and regulations
General, 75-0825
Lipids/steroids/sterols
Animals/experimental, 75-1084 75-1085
Human, 75-0815
Liver
Animals/experimental, 75-0848 75-0850, 75-0884, 75-1127 75-1198, 75-1337, 75-1385 75-1411, 75-1433
In vitro, 75-0853, 75-1201
Metabolism
General, 75-1433
Insects, 75-1436
Microorganisms, 75-1354
Rat, 75-1130
Sharks/rays, 75-1350
Nervous system
Animals/experimental, 75-1127
Nucleic acids
Microorganisms, 75-1063
Reproduction/growth
Animals/experimental, 75-0848 75-1085
Residue removal
Plants, 75-0986
Water, 75-1257
Residues/air
General, 75-1250
Residues/food and feed
Total diet, 75-0785
Animal feed, 75-0773, 75-1030
Dairy products, 75-0760, 75-0763 75-1005, 75-1009, 75-1023 75-1025, 75-1296
Meat, 75-0770
Poultry, 75-1261
Residues/humans
Adipose, 75-1262, 75-1289
Blood, 75-1022, 75-1258
Milk, 75-0760, 75-1288, 75-1289
Residues/non-target organisms
Amphibians, 75-1259
Birds, 75-1259
Crustacea, 75-1008, 75-1279
Fish, 75-1008, 75-1032, 75-1251
Reptiles, 75-1259
DDT (cont'd)
Residues/plants
Medicinals and condiments 75-0803
Residues/soil
General, 75-1030
Adsorption, 75-1253
Residues/water
Groundwater/rain, 75-1266
Oceans/seas, 75-1252, 75-1253
Respiration, cellular
Animals/experimental, 75-0869 75-0870
Spectrometry
Colorimetry, 75-1461
UV, 75-1452
Spinal cord
Animals/experimental, 75-0851
Therapeutic use
Cancer, 75-1197
Cushing's syndrome, 75-1197
Toxicity/experimental animals
Crustacea, 75-1166
Fish, 75-0911
Microorganisms, 75-0864, 75-0937
Plankton/algae, 75-0908
Sharks/rays, 75-1350
Toxicity/humans
Occupational, 75-0812, 75-0815

DDT derived compounds
see also Organochlorines
Biotransformation
Insects, 75-1434
Chromatography
Column, 75-0973
Enzyme activity
Aliesterase, 75-1434
Mixed function oxidases, 75-1434
Excretion
Fish, 75-1086
In vitro, 75-1069, 75-1086
Factors influencing metabolism/ toxicity
Adaptation, 75-1434
Lipids/steroids/sterols
Animals/experimental, 75-1152
Toxicity/experimental animals
General, 75-0910

DDT isomers
see Organochlorines

DDVP
see Dichlorvos

DEF
Biochemical effects
Animals/experimental, 75-1169
Enzyme activity
Animals/experimental, 75-1169
Mixed function oxidases, 75-1169

Demeton
see also Insecticides; Organophosphates
Factors influencing metabolism/ toxicity
Molecular structure, 75-0859
Residues/humans

Demeton (cont'd)
Urine, 75-1329
Toxicity/humans
Occupational, 75-1329

DFP
Behavior
Animals/experimental, 75-1176
Blood pressure
Animals/experimental, 75-0860
Brain
Animals/experimental, 75-1333
Enzyme activity
General, 75-1176
Cathepsin A, 75-0899
Cholinesterase, 75-1068, 75-1333
β-Glucuronidase, 75-1099
Immunology
In vitro, 75-1384
Vision
Animals/experimental, 75-1333

Diazinon
see also Organophosphates
Biotransformation
Human, 75-1056
Metabolism
Plankton/algae, 75-1341
Toxicity/humans
Intentional, 75-1056
Occupational, 75-1047

Dicamba
see also Herbicides
Photodecomposition, 75-0761
Residues/water
Rivers/streams, 75-1256
Spectrometry
Radiometry, 75-0991

Dichlobenil
see also Herbicides
Residues/soil
General, 75-0795

Dichlone
see also Organophosphates
Cell membranes
In vitro, 75-0878
Enzyme activity
ATPase, 75-0878

Dichloroaniline
Biotransformation
In vitro, 75-1339
Liver
In vitro, 75-1339

3,4-Dichloroaniline
see also Herbicides
Residues/food and feed
Cereals, 75-0793

Dichlorvos
see also Organophosphates
Amino acids/peptides/proteins
In vitro, 75-0875
Respiration, cellular, 75-1093
Biochemical effects
Microorganisms, 75-1199
Enzyme activity
Cholinesterase, 75-0842, 75-1081 75-1191, 75-1327
Cytochrome oxidase, 75-1404
Mixed function oxidases, 75-1093
Factors influencing metabolism/ toxicity
Humidity, 75-1081
Sex, 75-1064
Growth
Microorganisms, 75-1199
Plants, 75-1199
Lipids/steroids/sterols
In vitro, 75-1088
Liver
Animals/experimental, 75-1093
Mutagenesis/teratogenesis
General, 75-0832
Neonate
Animals/experimental, 75-0824
Nervous system
Animals/experimental, 75-1081
Respiration, cellular
Microorganisms, 75-1203
Spectrometry
Colorimetry, 75-0966, 75-1491 75-1493
Toxicity/experimental animals
Fish, 75-0842
Pig, 75-0824
Toxicity/humans
General, 75-0832
Occupational, 75-1327

Dichlozoline
Absorption
Rabbit, 75-1145
Blood/body fluids
Animals/experimental, 75-1145
Enzyme activity
Cholinesterase, 75-1145
Skin
Animals/experimental, 75-1145

Dicofol
Enzyme activity
Cholinesterase, 75-1142
Factors influencing metabolism/ toxicity
Interactions, 75-1142
Kidney
Animals/experimental, 75-1142

Dicrotophos
Biotransformation
In vitro, 75-0927

Dieldrin
see also Organochlorines
Amino acids/peptides/proteins
In vitro, 75-1193
Behavior
Animals/experimental, 75-1210
Catecholamines
Animals/experimental, 75-0940
Chromatography
Thin-layer, 75-1216
Cytological effects
Animals/experimental, 75-1391

Dieldrin (cont'd)
Digestive system
Animals/experimental, 75-0919
Distribution/storage
Duck, 75-1386
Mouse, 75-1401, 75-1402
Enzyme activity
Mixed function oxidases, 75-0848 75-0909, 75-0927
Excretion
Duck, 75-0865
Factors influencing metabolism/ toxicity
Formulation, 75-1386
Taxon, 75-1193, 75-1210
Glands
Animals/non-target, 75-0865
Immunology
Animals/experimental, 75-1386
Lipids/steroids/sterols
Animals/experimental, 75-1133
In vitro, 75-1133
Liver
Animals/experimental, 75-0848 75-0909, 75-1386
Nervous system
Animals/experimental, 75-1391 75-1403
Reproduction/growth
Animals/experimental, 75-0848 75-0909
Reproductive organs, male
Animals/experimental, 75-1133
Residues/food and feed
Total diet, 75-0785
Cereals, 75-0776
Dairy products, 75-0760, 75-1005 75-1009
Meat, 75-0770
Residues/humans
Milk, 75-0760
Residues/non-target organisms
General, 75-0799
Fish, 75-1251
Spectrometry
Radiometry, 75-1487
Toxicity/experimental animals
Fish, 75-0911
Guinea pig, 75-1391, 75-1403
Toxicity/non-target organisms
Birds, 75-1113

Difenamid
see Herbicides

Dihydrochlordene dicarboxylic acid
see also Organochlorines
Photodecomposition, 75-1026

***trans*-4,5-Dihydroxy-4,5-dihydroaldrin**
Metabolism
General, 75-0879

Dimecron
see Phosphamidon

Dimefox
see Organophosphates

Dimethirimol
Titration, 75-1453

Dimethoate
see also Organophosphates
Biotransformation
In vitro, 75-0927
Liver
Animals/experimental, 75-0841
Residues/air
Industrial, 75-1054
Reticuloendothelial system
Animals/experimental, 75-0841
Spectrometry
UV, 75-1465
Toxicity/experimental animals
Rat, 75-1380
Toxicity/humans
Occupational, 75-1054

***O,O*-Dimethyl 2,2-dichloro 1-hydroxyethyl phosphonate**
see NS 2662

Dinitramine
see Nitro compounds

Dinitrophenol
Biochemical effects
General, 75-0882

Diothyl
Photodecomposition, 75-0787

Dioxacarb
Biotransformation
Microorganisms, 75-0913

Dioxins
Behavior
Human, 75-1306
Blood/body fluids
Animals/experimental, 75-1437
Carcinogenesis
Animals/experimental, 75-1344
Chromatography
General, 75-0992
Cytological effects
Animals/experimental, 75-1344
Enzyme activity
ATPase, 75-1344
Mixed function oxidases, 75-0857
p-Nitrophenol glucuronyltransferase, 75-1071
UDP glucuronyltransferase 75-0856
Excretory system
Animals/experimental, 75-1376
Factors influencing metabolism/toxicity
Sex, 75-1071, 75-1376
Growth
Animals/experimental, 75-1376
Lipids/steroids/sterols
Animals/experimental, 75-1071
Human, 75-1306
Liver
Animals/experimental, 75-0857 75-1344, 75-1376, 75-1437
Skin
Human, 75-1306

Dioxins (cont'd)
Toxicity/experimental animals
Mouse, 75-1437
Toxicity/humans
Occupational, 75-1306

Diphenamid
Biochemical effects
Microorganisms, 75-0934

Dipterex
see Trichlorfon

Diquat
see also Herbicides
Enzyme activity
Cholinesterase, 75-1077
Lung
Animals/experimental, 75-1077
Residues/soil
Adsorption, 75-0802
Toxicity/experimental animals
Crustacea, 75-1357

Disulfoton
see also Organophosphates
Cytological effects
Animals/experimental, 75-1338
Muscle, striated
Animals/experimental, 75-1381
Vision
Animals/experimental, 75-1338 75-1381

Diuron
see also Herbicides
Biotransformation
Microorganisms, 75-1206
Enzyme activity
General, 75-1420, 75-1422
Liver
Animals/experimental, 75-1422
Residues/soil
General, 75-0795
Toxicity/experimental animals
Plants, 75-0897

DMDT
see Methoxychlor

DNBP
Chromatography
Thin-layer, 75-1460
Enzyme activity
Succinic dehydrogenase, 75-1369
Liver
Animals/experimental, 75-1369
Toxicity/experimental animals
Rat, 75-1369

DNOC
Chromatography
Thin-layer, 75-1460

Dowco 214
Respiration, cellular
Microorganisms, 75-1203

Dowco 217
Respiration, cellular
Microorganisms, 75-1203

2,4-DP
Chromatography
Thin-layer, 75-1460

Dyfonate
see also Organophosphates
Biotransformation
Microorganisms, 75-1123
Factors influencing metabolism/toxicity
Nutritional state, 75-1123
pH, 75-1123
Temperature, 75-1123
Residue dynamics, 75-0820

Ekatin
see Thiometon

Endosulfan
see also Organochlorines
Chromatography
Gas-liquid, 75-1454
Residue removal
Plants, 75-0986
Residues/food and feed
Dairy products, 75-1009
Toxicity/experimental animals
General, 75-1389
Fish, 75-0911

Endrin
see also Organochlorines
Biotransformation
Mouse, 75-1061
Rat, 75-0880
Factors influencing metabolism/toxicity
Taxon, 75-1061

EPTC
Biotransformation
In vitro, 75-1192
Mouse, 75-1192

Ethion
Chromatography
Gas-liquid, 75-1468
Enzyme activity
Cholinesterase, 75-0823
Residue degradation
Soil, 75-1003
Toxicity/humans
Occupational, 75-0823

Ethion derived compounds
Chromatography
Gas-liquid, 75-1468

Ethoprop
Toxicity/experimental animals
Microorganisms, 75-0873

Ethyl formate
see Fumigants

Ethyl thiometon
see Disulfoton

Ethylene dibromide
Residues/food and feed
Fruits, 75-0783

Ethylene oxide
Metabolism
Mouse, 75-1395
Toxicity/humans
Occupational, 75-1395

Fenitrothion
see also Organophosphates
Behavior
Animals/experimental, 75-1163 75-1164
Chromatography
Column, 75-1455
Enzyme activity
Cholinesterase, 75-0942, 75-1190
Residues/food and feed
General, 75-1248
Fruits, 75-1286
Toxicity/experimental animals
Fish, 75-1163, 75-1164
Vision
Animals/experimental, 75-0942 75-1190

Fenitrothion derived compounds
Chromatography
Gas-liquid, 75-0990

Fensulfothion
Residue degradation
Soil, 75-0765
Residues/food and feed
Vegetables, 75-0765
Respiration, cellular
Microorganisms, 75-1203

Fenthion
see also Organophosphates
Bioassay, 75-1224
Biotransformation
Plants, 75-1417

Fenuron
Adrenal
Animals/experimental, 75-1171
Amino acids/peptides/proteins
Animals/experimental, 75-1171
Blood cells
Animals/experimental, 75-1171
Kidney
Animals/experimental, 75-1171
Liver
Animals/experimental, 75-1171
Myocardium
Animals/experimental, 75-1171
Residues/soil
Adsorption, 75-1024
Respiratory system
Animals/experimental, 75-1171
Spleen
Animals/experimental, 75-1171
Thyroid
Animals/experimental, 75-1171

Ferbam
Metabolism
Rat, 75-1073

Fluenethyl
see also Insecticides
Enzyme activity

Fluenethyl (cont'd)
General, 75-1353
Toxicity/experimental animals
Insects, 75-1353
Mouse, 75-1353

Fluometuron
see also Fluorine Compounds
Spectrometry
UV, 75-1484
Toxicity/experimental animals
Plants, 75-0897

Fluorine Compounds
see also Fluometuron; SAN 6706; SAN 9789; TFM; Trifluralin
Carbohydrates
Animals/experimental, 75-1187
Respiration, cellular
In vitro, 75-1186

Folpet
see also Fungicides
Mutagenesis/teratogenesis
Animals/experimental, 75-1174

Formetanate
see also Insecticides
Enzyme activity
Cholinesterase, 75-1366
Kidney
Rat, 75-1366
Liver
Rat, 75-1366
Lung
Rat, 75-1366
Toxicity/experimental animals
Rat, 75-1366

Formothion
see also Organophosphates
Chromatography
Gas-liquid, 75-0978
Paper, 75-0978
Factors influencing metabolism/toxicity
Interactions, 75-1052
Toxicity/humans
Occupational, 75-1052

Foschlor
see Trichlorfon

Fosfotox R
see Dimethoate

Fospirate
see also Organophosphates
Factors influencing metabolism/toxicity
Sex, 75-1079

Fumigants
see Carbon tetrachloride; D-D; Ethyl formate; Ethylene dibromide; Ethylene dichloride; Ethylene oxide; Hexachlorobutadiene; Methyl bromide; Phosphine

Fungicides
see also Azide; Benomyl; Bordeaux mixture; Captafol; Captan; Copper; DNOC; Folpet; Hexachlorobenzene; Hymexazol; Maneb; Norobiocin; PCNB; Sulfur; Thiram; Trichlorophenol; Triphenyltin; Zineb; Ziram
Biochemical effects
Microorganisms, 75-1205
Chromatography
Gas-liquid, 75-1220, 75-1471
Electrolytes
Microorganisms, 75-1205
Residues/food and feed
Vegetables, 75-0804
Residues/soil
Movement, 75-1021
Reviews
Monitoring and residues, 75-0804

Granosan
see also Mercurials
Factors influencing metabolism/toxicity
Interactions, 75-1052
Prevention
Protective equipment, 75-1051
Toxicity/humans
Occupational, 75-1052

Granulosis virus
Residues/plants
Forest, 75-0797

Heptachlor
see also Insecticides; Organochlorines
Biotransformation
Plants, 75-1349
Chromatography
General, 75-1232
Thin-layer, 75-1234
Electrometry
Polarography, 75-1232, 75-1234
Residues/food and feed
Total diet, 75-0785
Dairy products, 75-1009
Residues/non-target organisms
General, 75-0799
Toxicity/experimental animals
Mouse, 75-1346

Heptachlor epoxide
see also Organochlorines
Chromatography
General, 75-1232
Thin-layer, 75-1234
Electrometry
Polarography, 75-1232, 75-1234
Residues/food and feed
Total diet, 75-0785
Dairy products, 75-0760, 75-1009
Residues/humans
Milk, 75-0760

Heptachlor epoxide (cont'd)
Residues/non-target organisms
General, 75-0799

Herbicides
see also Acrolein; Agallol; Alachlor; Amitrole; Aniline; Atrazine; Benthiocarb; Butylate; Chloramben; Chlorpropham; Cisanilide; Cyanazine; Cyprozin; Dalapon; DCPA; 3,4-Dichloroaniline; Difenamid; Diquat; Diuron; Ioxynil; Isopropalin; Linuron; Maleic hydrazide; MCPA; Methazole;3-methoxy-4,6 *bis*(isopropylamino)*s*-triazine; Metribuzin; Molinate;N-*sec*-butyl-4-*tert*-butyl-2,6-dinitroaniline; 1-Naphthaleneacetic acid; Naptalam; Nitrofen; Norflurazon; Paraquat; PCP; Pebulate; Phenmedipham; Picloram; Prometone; Prometryne; Propazine; Propham; Siduron; Simazine; Sodium arsenite; 2,4,5-T; Terbacil; Terbutryne; Triazines; Trifluralin; Vernolate
Biotransformation
Microorganisms, 75-1196
Chromatography
Gas-liquid, 75-1220, 75-1469
Economics, 75-1237, 75-1242
Factors influencing metabolism/toxicity
Adaptation, 75-1356
Molecular structure, 75-1196
pH, 75-0930
Taxon, 75-1196
Metabolism
General, 75-1428
Microorganisms, 75-0930
Residue removal
Water, 75-1265
Residues/plants
Medicinals and condiments 75-0803
Residues/soil
General, 75-0789
Movement, 75-0790
Reviews
Analysis, 75-0967
Spectrometry
Colorimetry, 75-1212
Toxicity/experimental animals
Fish, 75-1356
Toxicity/humans
Accidental, 75-0792
Toxicity/non-target organisms
General, 75-0792

Hexachlorobenzene
see also Fungicides
Analysis
Sample preparation, 75-0980 75-1215
Biochemical effects
Animals/experimental, 75-1153
Electrolytes
Animals/experimental, 75-1150
Enzyme activity
General, 75-1153, 75-1375
Lactic dehydrogenase, 75-1270
Mixed function oxidases, 75-0884 75-1131
Factors influencing metabolism/toxicity
Sex, 75-1131, 75-1270
Taxon, 75-0886
Liver
Animals/experimental, 75-1150
Metabolism
Rat, 75-1360, 75-1375
Morbidity and mortality statistics
USA-Louisiana, 75-0762
Porphyrins
General, 75-0886
Animals/experimental, 75-1131 75-1135, 75-1137, 75-1150 75-1151, 75-1375
Human, 75-1270
Residues/food and feed
Meat, 75-1271
Poultry, 75-1271
Residues/humans
General, 75-0762
Blood, 75-1270
Reviews
Toxicology and pharmacology 75-1137

Hexachlorobutadiene
see also Fumigants
Analysis
Sample preparation, 75-1215

Hydrogen cyanide
Biochemical effects
General, 75-0882

3-Hydroxy-5-methylisoxazol
see Hymexazole

Hydroxyisoxazole
Metabolism
Rat, 75-1101

Hymexazol
see also Fungicides
Metabolism
Plankton/algae, 75-1398
Toxicity/experimental animals
Fish, 75-1398
Mouse, 75-1398
Rat, 75-1398

Intration
see Thiometon

Ioxynil
see also Herbicides
Biotransformation
Microorganisms, 75-1020

Irradiation
Chromosomes/genes
Human, 75-1102

Isopropalin
see Nitro compounds

Juvenile hormones and analogs
see also Methoprene
Biotransformation
Insects, 75-0926

Kepone
see Chlordecone

Largon
see Fluorine compounds

Lasso
see Alachlor

Lead
see also Heavy metals
Residues/food and feed
General, 75-1284
Fruits, 75-1286
Meat, 75-0770
Toxicity/humans
Occupational, 75-0816

Lead arsenate
see Arsenicals

Leptophos
see also Organophosphates
Brain
Animals/experimental, 75-1096
Demyelination
Animals/experimental, 75-1096
Distribution/storage
Rat, 75-0780
Enzyme activity
Cholinesterase, 75-1096
Peripheral nerves
Animals/experimental, 75-1096
Residues/food and feed
General, 75-0782
Vegetables, 75-1040
Residues/plants
Tobacco, 75-0780
Spinal cord
Animals/experimental, 75-1096

Lindane
see also Organochlorines
Absorption
Rat, 75-1367
Amino acids/peptides/proteins
Human, 75-0815
Analysis
Sample preparation, 75-0980
Carcinogenesis
Animals/experimental, 75-1442
Human, 75-0812
Chromatography
General, 75-1232
Thin-layer, 75-1216

Lindane (cont'd)
Electrometry
Polarography, 75-1232
Enzyme activity
5-Aminolevulinate synthase 75-1149
Mixed function oxidases, 75-1406
Succinic dehydrogenase, 75-1369
Factors influencing metabolism/toxicity
Formulation, 75-1367
Interactions, 75-1136, 75-1149
Taxon, 75-1149
Lipids/steroids/sterols
Human, 75-0815
Liver
Animals/experimental, 75-1149 75-1369, 75-1406
Porphyrins
Animals/experimental, 75-1136
Residues/food and feed
Total diet, 75-0785
Dairy products, 75-1023
Toxicity/experimental animals
Rat, 75-1369
Toxicity/humans
Occupational, 75-0812, 75-0815 75-1055
Treatment of poisoning
General, 75-1055

Linuron
see also Herbicides
Bioassay, 75-1488
Chromatography
Thin-layer, 75-1492
Enzyme activity
General, 75-1420, 75-1422 75-1425
Growth
Microorganisms, 75-1393
Liver
Animals/experimental, 75-1422 75-1425
Residue degradation
Food and feed, 75-0788
Residues/soil
Adsorption, 75-1006

M-1704
Carbohydrates
Animals/experimental, 75-0920
Lipids/steroids/sterols
Animals/experimental, 75-0920

Malaoxon
see also Organophosphates
Chromatography
Thin-layer, 75-1230
Enzyme activity
Cholinesterase, 75-0905

Malathion
see also Organophosphates
Biochemical effects
Microorganisms, 75-1199

Malathion (cont'd)
Biotransformation
Microorganisms, 75-1134
Chromatography
Thin-layer, 75-1230
Cytological effects
Animals/experimental, 75-1421 75-1424
Enzyme activity
General, 75-0877, 75-1421 75-1424
Cholinesterase, 75-0905, 75-1155
Mixed function oxidases, 75-0933
Growth
Animals/experimental, 75-1416
Microorganisms, 75-1199
Plants, 75-1199
Liver
Animals/experimental, 75-0933 75-1421, 75-1424
In vitro, 75-0877
Respiration, cellular
Animals/experimental, 75-1424
Microorganisms, 75-1203
Spectrometry
Colorimetry, 75-0966
Toxicity/experimental animals
Amphibians, 75-1416
Fish, 75-0911
Microorganisms, 75-1413
Rat, 75-1380

Maleic hydrazide
Chromatography
Column, 75-0984
Gas-liquid, 75-0984

MCPA
see also Herbicides
Blood cells
Human, 75-1302
Chromatography
Thin-layer, 75-1460
EEG
Human, 75-1325
Enzyme activity
Cytochrome oxidase, 75-1404
Mutagenesis/teratogenesis
General, 75-1202
Spectrometry
Colorimetry, 75-1459
Toxicity/experimental animals
Rat, 75-1443
Toxicity/humans
Occupational, 75-1302, 75-1325
Treatment of poisoning
General, 75-1302

MCPP
Chromatography
Thin-layer, 75-1460
Toxicity/experimental animals
Rabbit, 75-1443

Mercurials
see also Granosan
Absorption

Mercurials (cont'd)
Fish, 75-0916
Amino acids/peptides/proteins
In vitro, 75-1115
Biotransformation
General, 75-1358
Blood/body fluids
In vitro, 75-1427
Brain
Human, 75-1000
Carbohydrates
Animals/experimental, 75-1187
Chromatography
Thin-layer, 75-1485, 75-1486
Digestive system
Animals/experimental, 75-0919
Environmental pollution, 75-1000 75-1001
Enzyme activity
Catalase, 75-1427
Excretion
Human, 75-1000
Mouse, 75-1388
Rat, 75-1388
Factors influencing metabolism/toxicity
Interactions, 75-0946, 75-1388
Temperature, 75-0916
Liver
In vitro, 75-1427
Mitosis/meiosis
In vitro, 75-1115
Nucleic acids
In vitro, 75-1115
Placental transfer
Animals/experimental, 75-1324
Human, 75-1324
Residues/food and feed
Cereals, 75-0776
Meat, 75-0770
Residues/humans
Urine, 75-1329
Residues/non-target organisms
Birds, 75-0774, 75-1031
Eggs, 75-0774
Residues/soil
General, 75-1019
Respiration, cellular
In vitro, 75-1186
Reviews
General, 75-1001
Analysis, 75-1001
Toxicology and pharmacology 75-1358
Safety standards
General, 75-1000
Toxicity/humans
Accidental, 75-0836, 75-1324 75-1328
Occupational, 75-0816, 75-1329
Toxicity/non-target organisms
Cow, 75-0840
Treatment of poisoning
α-Lipoic acid, 75-1427

Metaldehyde
Reproduction/growth
Animals/experimental, 75-1444

Metaldehyde (cont'd)
Toxicity/experimental animals
Rat, 75-1444

Methabenzthiazuron
Chromatography
Thin-layer, 75-1221

Methiocarb
Biotransformation
In vitro, 75-0885
Embryo/fetus
In vitro, 75-0885

Methoprene
see also Juvenile hormones and analogs
Biotransformation
Plants, 75-1158
Chromatography
Gas-liquid, 75-0983
Photodecomposition, 75-1275
Methoprene, 75-1274

3-Methoxy-4,6 *bis*(isopropylamino)*s*-triazine
see also Herbicides; Triazines
Toxicity/experimental animals
Microorganisms, 75-0937

Methoxychlor
see also Organochlorines
Metabolism
Plankton/algae, 75-1341
Residues/food and feed
Fruits, 75-1294
Toxicity/experimental animals
Microorganisms, 75-0937

Methoxychlor derived compounds
Metabolism
General, 75-0910
Toxicity/experimental animals
General, 75-0910

Methyl bromide
Chromatography
Gas-liquid, 75-1225
Residues/food and feed
Cereals, 75-1013
Safety standards
General, 75-1311
Toxicity/humans
Occupational, 75-1311

2-Methyl-4-chlorophenoxyacetic acid
see MCPA

Methyl demeton
Toxicity/experimental animals
Microorganisms, 75-0937

Methyl parathion
see also Organophosphates
Amino acids/peptides/proteins
Animals/experimental, 75-0843
Chromatography
Gas-liquid, 75-1483
Enzyme activity
Cholinesterase, 75-0863
Factors influencing metabolism/toxicity
Taxon, 75-0863
Feathers
Animals/experimental, 75-0843
Muscle, striated
Animals/experimental, 75-0843
Residues/air
Industrial, 75-1054
Toxicity/humans
Occupational, 75-1054

Methylmercuric chloride
Factors influencing metabolism/toxicity
Molecular structure, 75-1132
Metabolism
Rat, 75-1132

Metribuzin
see Herbicides

Mevinphos
see also Organophosphates
Distribution/storage
Human, 75-1044
Enzyme activity
Cholinesterase, 75-1044
Toxicity/humans
Intentional, 75-1044

Mexacarbate
Biotransformation
In vitro, 75-0885
Embryo/fetus
In vitro, 75-0885

Milbex
Brain
Animals/experimental, 75-1143
Enzyme activity
Cytochrome oxidase, 75-1143
Succinate dehydrogenase, 75-1143
Safety standards
TLV/MAC, 75-1143

Mirex
see also Organochlorines
Cytochromes
Animals/experimental, 75-1433
Distribution/storage
Rat, 75-0780
Eggshell effects
Animals/experimental, 75-1062 75-1103
Enzyme activity
Lactic dehydrogenase, 75-1087
Mixed function oxidases, 75-1062 75-1433
Experimental design
Toxicology and pharmacology 75-1343
Liver
Animals/experimental, 75-1062 75-1433
Metabolism
General, 75-1433
Residues/non-target organisms
General, 75-1034, 75-1035
Residues/plants
General, 75-1036

Mirex (cont'd)
Residues/soil
General, 75-1036
Residues/water
Lakes/ponds, 75-1036
Toxicity/experimental animals
Fish, 75-1209

MNFA
Enzyme activity
General, 75-1370
Growth
Microorganisms, 75-1370

Molinate
see also Herbicides
Biotransformation
In vitro, 75-1192
Mouse, 75-1192

Molluscicides
Biochemical effects
Animals/experimental, 75-1441
Toxicity/experimental animals
Rat, 75-1441

Monuron
Biotransformation
Microorganisms, 75-1206
Chromatography
Thin-layer, 75-1492
Residues/soil
Adsorption, 75-1024

Morfamquat
Enzyme activity
Cholinesterase, 75-1077
Lung
Animals/experimental, 75-1077

Naled
see also Organophosphates
Enzyme activity
Cholinesterase, 75-0823, 75-0950
Respiration, cellular
Microorganisms, 75-1203
Toxicity/experimental animals
Fish, 75-0950
Toxicity/humans
Occupational, 75-0823

1-Naphthaleneacetic acid
see also Herbicides
Spectrometry
Fluorometry, 75-1218

1-Naphthol
Biotransformation
Microorganisms, 75-0894

α-Naphthyl isothiocyanate
Enzyme activity
Mixed function oxidases, 75-0876
Liver
In vitro, 75-0876

Naptalam
see also Herbicides
Chromatography
Thin-layer, 75-1221

Neguvon
see Trichlorfon

Neodecanoic acid
Biotransformation
Plants, 75-1159

Nitro compounds
see also Dinitramine; DNOC; Isopropalin; Nitrofen; PCNB; Trifluralin
Biotransformation
In vitro, 75-1098

Nitrofen
see also Herbicides
Photodecomposition, 75-0777 75-1273

Norbormide
see also Rodenticides
Carbohydrates
Animals/experimental, 75-0891
Factors influencing metabolism/toxicity
Nutritional state, 75-0891

Norflurazon
see also Herbicides
Biotransformation
Plants, 75-0955
Distribution/storage
Plants, 75-0955

Novobiocin
see also Fungicides
Liver
Human, 75-1308

NS 2662
see also Organophosphates
Enzyme activity
ATPase, 75-1400
Toxicity/experimental animals
Mouse, 75-1400

Organochlorines
see also Aldrin; BHC; BHC isomers; Chlordane; DDE; DDT; DDT derived compounds; DDT isomers; Dicofol; Dieldrin; Dihydrochlordene dicarboxylic acid; Endosulfan; Endrin; Heptachlor; Heptachlor epoxide; Lindane; Methoxychlor; Mirex; Photoaldrin; TDE; Toxaphene
Analysis
General, 75-1228
Bioassay, 75-1229
Chromatography
Column, 75-0974
Gas-liquid, 75-1010, 75-1213 75-1217, 75-1451, 75-1472

Organochlorines (cont'd)
Gas-liquid, 75-1474, 75-1480
Thin-layer, 75-1475, 75-1485 75-1486
Digestive system
In vitro, 75-1405
Distribution/storage
Rat, 75-1313
Eggshell effects
Animals/non-target, 75-0768
Electrometry
Amperometry, 75-1482
Environmental pollution
Organochlorines, 75-1264
Enzyme activity
Cholinesterase, 75-1315, 75-1316
Pepsin, 75-1405
Factors influencing metabolism/toxicity
Adaptation, 75-1347, 75-1435
Age, 75-1183
Interactions, 75-0936
Liver
Animals/experimental, 75-1347
Neonate
Animals/experimental, 75-1313
Photodecomposition, 75-1301
Placental transfer
Animals/experimental, 75-1313
Reproduction/growth
Animals/experimental, 75-1183
Human, 75-1313
Residue degradation
General, 75-1293
Residue removal
3cow, 75-1023
Water, 75-1265
Residues/food and feed
General, 75-1269, 75-1284
Total diet, 75-1292
Animal feed, 75-1280
Dairy products, 75-1298
Fruits, 75-1298
Meat, 75-1263, 75-1272, 75-1298
Vegetables, 75-1298
Residues/humans
General, 75-1037
Adipose, 75-0763, 75-1290 75-1297
Blood, 75-1283, 75-1313
Milk, 75-1284, 75-1297
Residues/non-target organisms
Birds, 75-0772, 75-0774, 75-1010 75-1297, 75-1299
Crustacea, 75-0786, 75-1249
Eggs, 75-0774, 75-1297, 75-1300
Fish, 75-0786, 75-1010, 75-1249 75-1299
Molluscs, 75-1010, 75-1249
Pig, 75-1298
Reptiles, 75-0772
Shrew, 75-0772
Wolf, 75-1038
Residues/plants
Tobacco, 75-0780
Residues/soil
Movement, 75-1295

Organochlorines (cont'd)
Residues/water
General, 75-0801, 75-1291 75-1299
Oceans/seas, 75-0786
Spectrometry
Colorimetry, 75-1477
Radiometry, 75-1229
Titration, 75-1482
Toxicity/experimental animals
General, 75-1301
Fish, 75-1183, 75-1345
Microorganisms, 75-1182
Toxicity/humans
Occupational, 75-0816, 75-1315 75-1316
Toxicity/non-target organisms
Birds, 75-0768
Eggs, 75-0768

Organophosphates
see also Azinphosmethyl; Chlorfenvinphos; Chlorpyrifos; Cruformate; DEF; Demeton; DFP; Dichlone; Dichlorvos; Dimethoate; Dioxathion; Dyfonate; Ethion; Fenitrothion; Fospirate Leptophos; Malathion; Malaxon; Mevinphos; Naled; NS 2662; Paraoxon; Parathion; Phorate; Phosmet; Phospholan; Pyridafenthion; R-16661; Ronnel; Salithion; Sarin; Soman; Thiometon; Trichlorfon
Analysis
General, 75-0968
Biotransformation
General, 75-0960
In vitro, 75-1120
Insects, 75-1434
Microorganisms, 75-1119
Blood cells
Animals/experimental, 75-1211
Brain
Animals/experimental, 75-1211
Carbohydrates
Animals/experimental, 75-1187
Carcinogenesis
Human, 75-0812
Cell membranes
In vitro, 75-0888, 75-0889
Chromatography
Column, 75-0993, 75-1222
Gas-liquid, 75-1213, 75-1469 75-1480
Thin-layer, 75-0994, 75-1475 75-1485, 75-1486, 75-1490
Chromosomes/genes
Human, 75-1046
Digestive system
In vitro, 75-1405
Enzyme activity
α-chymotrypsin, 75-1120
Aliesterase, 75-1434
Cholinesterase, 75-0817, 75-0822 75-0858, 75-0968, 75-1082 75-1116, 75-1211, 75-1315 75-1316, 75-1321

Organophosphates (cont'd)
Esterases, 75-1118, 75-1120
Mixed function oxidases, 75-1434
para-Nitrophenyl phosphatase 75-0888, 75-0889
Pepsin, 75-1405
Enzyme assay
Cholinesterase, 75-1456, 75-1494
Experimental design
Toxicology and pharmacology 75-0858
Factors influencing metabolism/toxicity
Adaptation, 75-1434, 75-1436
Interactions, 75-0936
Molecular structure, 75-0958 75-0959, 75-0961, 75-0963 75-1118
Liver
Human, 75-1308
Metabolism
General, 75-0959
Insects, 75-1436
Muscle, striated
Animals/experimental, 75-1082
Nervous system
Human, 75-1048
Peripheral nerves
Animals/experimental, 75-1082
Photodecomposition, 75-1282 75-1301
Residue removal
Water, 75-1265
Residues/food and feed
General, 75-1269
Residues/plants
Medicinals and condiments 75-0803
Respiration, cellular
In vitro, 75-1186
Reviews
Epidemiology, prevention, and treatment, 75-0817
Toxicology and pharmacology 75-0959, 75-1157
Spectrometry
Colorimetry, 75-1494
Mass spectrometry, 75-0994
Radiometry, 75-0969
Toxicity/experimental animals
General, 75-0961, 75-0963 75-1301
Fish, 75-1345
Pheasant, 75-1387
Toxicity/humans
Accidental, 75-1304, 75-1305
Intentional, 75-1046
Occupational, 75-0812, 75-0816 75-0822, 75-1046, 75-1315 75-1316, 75-1321
Treatment of poisoning
Atropine, 75-1048
Oximes, 75-0817
Vision
Animals/experimental, 75-1188
Human, 75-1048, 75-1305

Oxamyl
see also Insecticides
Toxicity/humans
Accidental, 75-1304

Paraoxon
Blood cells
In vitro, 75-0855
Enzyme activity
Cholinesterase, 75-0905, 75-1138
Esterases, 75-0855
β-Glucuronidase, 75-1099
Factors influencing metabolism/toxicity
Disease state, 75-1138
Immunology
Animals/experimental, 75-1075

Paraquat
see also Herbicides
Alimentary tract
Human, 75-0828
Amino acids/peptides/proteins
Animals/experimental, 75-1173
Carcinogenesis
General, 75-0828
Catecholamines
Animals/experimental, 75-1076
In vitro, 75-1067
Embryo/fetus
Animals/experimental, 75-0871
Enzyme activity
Cholinesterase, 75-1077
Urease, 75-1154
Factors influencing metabolism/toxicity
Formulation, 75-1314
Growth
Microorganisms, 75-1154
Kidney
Human, 75-1045
Liver
Human, 75-1045
Lung
Animals/experimental, 75-0862 75-0917, 75-1077
Human, 75-0828, 75-0831 75-1045, 75-1059
Morbidity and mortality statistics
General, 75-0828
United Kingdom, 75-1067
Nucleic acids
Animals/experimental, 75-1173
Reproductive organs, female
Animals/experimental, 75-0871
Reproductive organs, male
Animals/experimental, 75-0871
Residues/soil
Adsorption, 75-0802, 75-0807
Safety standards
Toxicity rating class, 75-1314
Toxicity/experimental animals
Microorganisms, 75-1154
Toxicity/humans
General, 75-0831, 75-1045 75-1314

Paraquat (cont'd)
Accidental, 75-1059, 75-1319
Intentional, 75-0829, 75-1059 75-1318
Occupational, 75-0828
Treatment of poisoning
General, 75-0829, 75-0831
Adsorbents, 75-1317
Bentonite and purgatives, 75-0862
Superoxide dismutase and *d*-propranolol, 75-1067

Parathion
see also Organophosphates
Amino acids/peptides/proteins
In vitro, 75-1193
Behavior
Animals/experimental, 75-1210
Biotransformation
Microorganisms, 75-0907, 75-1141
Plants, 75-1378
Cell membranes
Animals/experimental, 75-1109
Distribution/storage
Rat, 75-1363
Electrolytes
In vitro, 75-1070
Enzyme activity
Cholinesterase, 75-0863, 75-0905 75-1080, 75-1104, 75-1109 75-1175
Factors influencing metabolism/toxicity
Age, 75-1080, 75-1175
Interactions, 75-1418
Sex, 75-1175
Taxon, 75-0863, 75-1193, 75-1210
Kidney
In vitro, 75-1070
Liver
Animals/experimental, 75-1109
Metabolism
Microorganisms, 75-1354
Rat, 75-1418
Morbidity and mortality statistics
Tunisia, 75-0830
Neonate
Animals/experimental, 75-1080
Prevention
Disposal, 75-0907
Residues/humans
Urine, 75-1329
Respiration, cellular
Animals/experimental, 75-1109
In vitro, 75-1092
Toxicity/experimental animals
Rat, 75-1418
Toxicity/humans
Accidental, 75-1304
Occupational, 75-1329

PCNB
see Fungicides; Nitro compounds

PCP
see also Herbicides
Biotransformation
Human, 75-1107
Microorganisms, 75-1128

PCP (cont'd)
Mouse, 75-1107
Rat, 75-1107
Chromatography
Gas-liquid, 75-0972
Thin-layer, 75-1464
Growth
Microorganisms, 75-1392
Residues/food and feed
Animal feed, 75-1014
Respiration, cellular
In vitro, 75-0861
Toxicity/experimental animals
Microorganisms, 75-0937
Toxicity/humans
Occupational, 75-1107

PCP derived compounds
Biotransformation
Microorganisms, 75-0872, 75-1177
Chromatography
Gas-liquid, 75-1223
Residues/food and feed
Poultry, 75-0872
Residues/non-target organisms
Chicken, 75-0779
Eggs, 75-0779

Pebulate
see also Herbicides
Biotransformation
In vitro, 75-1192
Mouse, 75-1192

Pepein S-50
see DDT

Phenamiphos
Biotransformation
General, 75-0962
Respiration, cellular
Microorganisms, 75-1203

Phencapton
Brain
Animals/experimental, 75-1185
Chromatography
Thin-layer, 75-1227

Phenmedipham
see also Herbicides
Chromatography
Thin-layer, 75-1233

Phenols
Chromatography
Gas-liquid, 75-1476

Phenothrin
Metabolism
In vitro, 75-0928
Rat, 75-0928

Phenylmercuric chloride
Factors influencing metabolism/ toxicity
Molecular structure, 75-1132
Metabolism
Rat, 75-1132

Phorate
see also Organophosphates

Phorate (cont'd)
Analysis
Sample preparation, 75-1470
Biotransformation
Helminths, 75-1184
Microorganisms, 75-1184
Chromatography
Gas-liquid, 75-1470

Phosalone
Enzyme activity
Cholinesterase, 75-0823
Residue degradation
Plants, 75-1003
Toxicity/humans
Occupational, 75-0823

Phosmet
Residues/food and feed
General, 75-1248

Phosphamidon
Biotransformation
In vitro, 75-0927
Chromatography
Gas-liquid, 75-1463
Chromosomes/genes
In vitro, 75-1397
Mutagenesis/teratogenesis
In vitro, 75-1397

Phosphorus
Toxicity/humans
Accidental, 75-0821
Intentional, 75-0821
Treatment of poisoning
General, 75-0821

Photodieldrin
Factors influencing metabolism/ toxicity
Biological magnification, 75-0847
Light/radiation, 75-1340
Taxon, 75-0847
Metabolism
General, 75-0847
Plankton/algae, 75-1340
Plants, 75-1340

Phoxim
Toxicity/experimental animals
Fish, 75-0922

Picloram
see also Herbicides
Residues/soil
General, 75-1039
Adsorption, 75-0767, 75-1011
Therapeutic use
Cancer, 75-1334

Piperonyl butoxide
see also Synergists
Enzyme activity
Mixed function oxidases, 75-0941 75-1179, 75-1201
Liver
In vitro, 75-1201
Mixed function oxidases, 75-0941

Pirimicarb
Titration, 75-1453

Plictran
Cardiovascular system
Animals/experimental, 75-0949
Factors influencing metabolism/ toxicity
Route, 75-0949
Respiratory system
Animals/experimental, 75-0949
Toxicity/experimental animals
Sheep, 75-0949

Polychlorinated biphenyls
Analysis
Sample preparation, 75-0980
Carcinogenesis
Animals/experimental, 75-0867
Chromatography
Column, 75-0973
Gas-liquid, 75-0972, 75-1217
Environmental pollution, 75-1244
Excretion
Mouse, 75-0943
Factors influencing metabolism/ toxicity
Interactions, 75-0864
Taxon, 75-0886
Placental transfer
Animals/experimental, 75-0943
Porphyrins
General, 75-0886
Residues/food and feed
Dairy products, 75-1005
Residues/humans
Blood, 75-1258
Residues/non-target organisms
Crustacea, 75-0786, 75-1008
Fish, 75-0786, 75-1008, 75-1032
Plankton/algae, 75-0775
Residues/water
Oceans/seas, 75-0786
Spectrometry
Colorimetry, 75-1477
Toxicity/experimental animals
Microorganisms, 75-0864, 75-1182
Plankton/algae, 75-0908

Polychloropinene
Liver
Animals/experimental, 75-1172
Porphyrins
Animals/experimental, 75-1172

Pralidoxime
see also Antidotes
Blood cells
Animals/experimental, 75-1390
Enzyme activity
General, 75-1390
Cholinesterase, 75-0939
Factors influencing metabolism/ toxicity
Nutritional state, 75-0939
pH, 75-0939

Pralidoxime (cont'd)
Heart
Animals/non-target, 75-1390

Prolan
Biotransformation
General, 75-1194

Promecarb
see also Carbamates
Biochemical effects
Animals/experimental, 75-1365
Kidney
Rat, 75-1365
Liver
Rat, 75-1365
Toxicity/experimental animals
Mouse, 75-1365
Rat, 75-1365

Prometone
see Herbicides; Triazines

Prometryne
see also Herbicides Triazines
Biochemical effects
Microorganisms, 75-0934
Biotransformation
Microorganisms, 75-0935
Residue degradation
Soil, 75-1254
Residues/soil
General, 75-1251, 75-1254
Movement, 75-1287
Toxicity/experimental animals
Plants, 75-0897

Propanil
Blood cells
Animals/experimental, 75-0953
Chromatography
Thin-layer, 75-0953
Kidney
Animals/experimental, 75-0953
Liver
Animals/experimental, 75-0953
Residues/soil
General, 75-0793

Propazine
see Herbicides; Triazines

Propham
see also Herbicides
Enzyme activity
Cholinesterase, 75-1327
Residue removal
Water, 75-1028
Residues/food and feed
Vegetables, 75-0764
Spectrometry
Infrared, 75-0995
Toxicity/humans
Occupational, 75-1327

Propoxur

Enzyme activity
Acetanilide hydroxylase, 75-1348

Propoxur (cont'd)
Aminopyrine *N*-demethylase
75-1348
Experimental design
Toxicology and pharmacology
75-1065
Lipids/steroids/sterols
Animals/experimental, 75-1348
Toxicity/experimental animals
General, 75-1065
Rat, 75-1348

Pyrazon
Chromatography
Gas-liquid, 75-0989
Metabolism
General, 75-1277
Residue degradation
Water, 75-1277
Residue dynamics, 75-1277
Residues/food and feed
Vegetables, 75-1285

Pyrethrins
see also Dimethrin
Biochemical effects
General, 75-1372
Biotransformation
Rat, 75-0896
Enzyme activity
General, 75-1372
Factors influencing metabolism/
toxicity
Molecular structure, 75-1016
Microsomes
In vitro, 75-1372
Photodecomposition, 75-1016
Spectrometry
Colorimetry, 75-0977
NMR, 75-0965
Toxicity/experimental animals
General, 75-1016

Pyridafenthion
see also Organophosphates
Chromatography
Gas-liquid, 75-1226
Thin-layer, 75-1226

Quinalphos
see also Insecticides
Spectrometry
Fluorometry, 75-0975

R-16661
see also Organophosphates
Biotransformation
General, 75-0958

Radosan
Residues/air
General, 75-1250

Repellents
see 4-Aminopyridine

Rodenticides
see Norbormide;5-*p*-Chlorophenyl silatrane; Sodium fluoride; Sodium fluoroacetate; Thallium; Warfarin

Rogor
see Dimethoate

Ronnel
see also Organophosphates
Spectrometry
Colorimetry, 75-0966

Rotenone
see also Botanicals
Biochemical effects
General, 75-1372
Chromatography
Thin-layer, 75-1221
Enzyme activity
General, 75-1372
Microsomes
In vitro, 75-1372
Photodecomposition, 75-1301
Spectrometry
Colorimetry, 75-0977
Toxicity/experimental animals
General, 75-1301

Salicylate
Factors influencing metabolism/
toxicity
Interactions, 75-0947
Metabolism
Rat, 75-0947

Salithion
see also Organophosphates
Biotransformation
Plants, 75-0852
Excretion
Rat, 75-0852

SAN 6706
see also Fluorine Compounds
Biotransformation
Plants, 75-0955
Distribution/storage
Plants, 75-0955
Metabolism
Plants, 75-0902

SAN 9789
see also Fluorine Compounds
Metabolism
Plants, 75-0902

Sarin
see also Organophosphates
Immunology
Animals/experimental, 75-1075
Vision
Human, 75-1139

Seed Dressing T
see Thiram

Sevin
see Carbaryl

Simazine
see also Herbicides; Triazines
Chromatography
Gas-liquid, 75-0976
Thin-layer, 75-1216
Residue degradation
Soil, 75-1254

Simazine (cont'd)
Residue removal
Water, 75-1027
Residues/food and feed
Cereals, 75-0766
Vegetables, 75-0766
Residues/soil
General, 75-0766, 75-0795
75-1254

Sodium arsenite
see Arsenicals; Herbicides

Sodium fluoride
see also Rodenticides
Biochemical effects
General, 75-0882

Sodium fluoroacetate
see also Rodenticides
Biochemical effects
General, 75-0882

Soman
see also Organophosphates
Enzyme activity
Cholinesterase, 75-1091
Immunology
Animals/experimental, 75-1075
Nucleic acids
Animals/experimental, 75-1091

Strychnine
Toxicity/non-target organisms
General, 75-1309

Sulfur
see Fungicides

Sumithion
see Fenitrothion

Synergists
see also Piperonyl butoxide
Cytochromes
Animals/experimental, 75-1433
Enzyme activity
Mixed function oxidases, 75-1433
Liver
Animals/experimental, 75-1433
Metabolism
General, 75-1433

2,4,5-T
see also Herbicides
Biochemical effects
General, 75-0904
Biotransformation
Rat, 75-1105
Chromatography
Gas-liquid, 75-1478
Electrolytes
Animals/experimental, 75-1083
Enzyme assay
Cholinesterase, 75-1478
Excretion
Rat, 75-1083
Excretory system
Animals/experimental, 75-1361
Factors influencing metabolism/toxicity
Age, 75-1361

2,4,5-T (cont'd)
Kidney
Animals/experimental, 75-0904
Lipids/steroids/sterols
In vitro, 75-0854
Liver
Animals/experimental, 75-0904
Metabolism
Dog, 75-1361
Rat, 75-1361
Residues/non-target organisms
General, 75-0798
Residues/plants
General, 75-0798
Residues/water
General, 75-0798
Spleen
Animals/experimental, 75-0904
Toxicity/experimental animals
Microorganisms, 75-0937

Tachigaren
see Hymexazol

2,4,5-TB
Biotransformation
Rat, 75-1106

TCA
Chromatography
Thin-layer, 75-1460
Residues/food and feed
General, 75-0781
Residues/water
Rivers/streams, 75-1018

TDE
see also Organochlorines
Analysis
Sample preparation, 75-0980
Biotransformation
Rat, 75-1129
Enzyme activity
Ornithine decarboxylase, 75-1094
Excretion
Rat, 75-1129
Neonate
Animals/experimental, 75-0925
Residue removal
Plants, 75-0986
Spectrometry
Colorimetry, 75-1461
Therapeutic use
Cancer, 75-0874, 75-1335
Cushing's syndrome, 75-1160

Tebuthiuron
see Herbicides

Tepa
see also Chemosterilants
Enzyme activity
Cholinesterase, 75-1114

Terbacil
see also Herbicides
Residues/soil
General, 75-0795

Terbutryne
see also Herbicides
Factors influencing metabolism/toxicity
Humidity, 75-0794
Temperature, 75-0794
Residues/soil
General, 75-0794
Toxicity/experimental animals
Plants, 75-0794

Tetrachlorvinphos
Enzyme activity
Cholinesterase, 75-1191
GOT, 75-1191
Spectrometry
Colorimetry, 75-0966

Tetramethylthiuram disulfide
see Thiram

TFM
Biotransformation
Fish, 75-0951
Residues/non-target organisms
General, 75-0906
Respiration, cellular
In vitro, 75-1351
Toxicity/experimental animals
General, 75-0906
Fish, 75-1351

Thallium
see also Rodenticides
Prevention
Marten, 75-0834
Toxicity/humans
General, 75-1060
Toxicity/non-target organisms
Badger, 75-0834
Fox, 75-0833
Treatment of poisoning
Prussian blue, 75-1060

Thio-tepa
Enzyme activity
Cholinesterase, 75-1108
Factors influencing metabolism/toxicity
Taxon, 75-1108
Lung
Animals/experimental, 75-1108

Thiometon
Blood cells
Animals/experimental, 75-1390
Chromatography
Thin-layer, 75-1227
Enzyme activity
General, 75-1390
Cholinesterase, 75-1364
Heart
Animals/non-target, 75-1390
Toxicity/experimental animals
Rat, 75-1364
Toxicity/humans
Occupational, 75-1364

Thioquinox
Chromatography
Thin-layer, 75-1221

Thiram
see also Fungicides
Biochemical effects
Animals/experimental, 75-1169
Microorganisms, 75-1156
Enzyme activity
GOT, 75-0890
Mixed function oxidases, 75-1169 75-1207
Factors influencing metabolism/ toxicity
Age, 75-1207
Growth
Animals/experimental, 75-0890
Liver
Animals/experimental, 75-0890 75-1169, 75-1207
Toxicity/experimental animals
Microorganisms, 75-1156
Toxicity/humans
Occupational, 75-1320

TMTD
see Thiram

Toxaphene
see also Organochlorines
Behavior
Animals/experimental, 75-1210
Biochemical effects
Animals/experimental, 75-1281
Biotransformation
Rat, 75-0895
Chromatography
Column, 75-1455
Chromosomes/genes
Human, 75-0839
Digestive system
Animals/experimental, 75-0919
Factors influencing metabolism/ toxicity
Taxon, 75-1210
Residue removal
Food and feed, 75-1281
Safety standards
TLV/MAC, 75-1281
Toxicity/experimental animals
Mouse, 75-1281
Rat, 75-1281
Toxicity/humans
Accidental, 75-0839
Toxicity/non-target organisms
Cow, 75-1330

Triazines
see also Ametryne; Atratone; Atrazine; Herbicides;3-methoxy-4,6 *bis*(isopropylamino)*s*-triazine; -Prometone; Prometyne; Propazine; Simazine
Chromatography
Gas-liquid, 75-1466, 75-1473
Thin-layer, 75-1479
Spectrometry
Fluorometry, 75-1479

Trichlorfon
see also Organophosphates
Biochemical effects
General, 75-1415
Microorganisms, 75-1199
Catecholamines
Animals/experimental, 75-1448
Chromosomes/genes
Animals/experimental, 75-1368
Cytological effects
Animals/experimental, 75-1415
Enzyme activity
General, 75-1415
Cholinesterase, 75-1117, 75-1142 75-1419, 75-1448
Factors influencing metabolism/ toxicity
Interactions, 75-1142
Sex, 75-1079
Growth
Animals/experimental, 75-1416
Microorganisms, 75-1199
Plants, 75-1199
Kidney
Animals/experimental, 75-1142
Liver
Animals/experimental, 75-0841 75-1415
Muscle, smooth
In vitro, 75-1332
Muscle, striated
In vitro, 75-1332
Mutagenesis/teratogenesis
Animals/experimental, 75-1112
Reproduction/growth
Animals/experimental, 75-1419
Residues/air
Industrial, 75-1054
Reticuloendothelial system
Animals/experimental, 75-0841
Spectrometry
Colorimetry, 75-1491
UV, 75-1465
Toxicity/experimental animals
Amphibians, 75-1416
Microorganisms, 75-0937, 75-1413
Toxicity/humans
Occupational, 75-1054

Trifluralin
see also Fluorine compounds; Herbicides; Nitro compounds
Biotransformation
Microorganisms, 75-0956
Photodecomposition, 75-1276
Residues/air
Rural, 75-1276
Residues/soil
Volatilization, 75-0778

Triphenyltin
see also Fungicides
Biotransformation
Plants, 75-0931
Rat, 75-0931

Tritox Liq 30
see DDT; Lindane; Methoxychlor

Vernolate
see also Herbicides
Biotransformation
In vitro, 75-1192
Mouse, 75-1192

Warfarin
see also Rodenticides
Amino acids/peptides/proteins
Animals/experimental, 75-0944
Chromatography
Thin-layer, 75-1221
Distribution/storage
Rat, 75-0912
Factors influencing metabolism/ toxicity
Interactions, 75-0947
Nutritional state, 75-0944
Liver
Animals/experimental, 75-0944
Metabolism
Rat, 75-0947

Zineb
see also Carbamates; Fungicides
Biochemical effects
Animals/experimental, 75-1189
Chromosomes/genes
Animals/experimental, 75-1368
Human, 75-1049
Enzyme activity
GOT, 75-0890
Growth
Animals/experimental, 75-0890 75-1189
Immunology
General, 75-1368
Liver
Animals/experimental, 75-0890
Lung
Animals/experimental, 75-1189
Mitosis/meiosis
Human, 75-1049
Reproduction/growth
Animals/experimental, 75-1368
Skin
Animals/experimental, 75-1189
Toxicity/humans
Occupational, 75-1049
Vision
Animals/experimental, 75-1189

Ziram
see also Carbamates; Fungicides
Biotransformation
Rat, 75-1362
Enzyme activity
Alkaline phosphatase, 75-1408
Reproduction/growth
Animals/experimental, 75-1368

Author Index

Abe, E., 75-1178
Abou-Donia, M.B., 75-1096
Abramova, E.L., 75-1484
Adamczewska, M., 75-1460
Adamovic, V., 75-0763
Adamovic, V.M., 75-1272
Adamson, R.H., 75-1008, 75-1432
Addison, R.F., 75-1352
Agnihotri, V.P., 75-0791, 75-1156
Ahlborg, U.G., 75-1107
Akeyama, N., 75-0920
Akimoto, T., 75-0773, 75-1280
Alam, M.T., 75-0881
Alexander, M., 75-0846
Alexandrescu, S., 75-1394
Alexandri, Al.V., 75-1394
Allen, J.G., 75-1198
Almeida, J.B., 75-1453
Alvarez-Cervera, J., 75-1335
Anderson, J.B., 75-1146, 75-1438
Andia, A.M.G., 75-1371
Ando, M., 75-1101
Anina, I.A., 75-1144
Annicchiarico Sebastiani, L., 75-1287
Antyukov, M.A., 75-0840
Aoi, H., 75-1412
Aoki, Z., 75-1412
Arai, M., 75-1412
Araki, M., 75-1381
Aravindakshan, A., 75-1161
Arestov, I.G., 75-0840
Armstrong, D.A., 75-1382
Arnold, D.W., 75-1174
Arnold, J.S., 75-0767
Arsenijevic, M., 75-0816
Asaka, S., 75-1225
Atallah, Y.H., 75-0780
Axelson, O., 75-0818, 75-0827
Azum-Gelade, M.C., 75-1344
Bachofer, R., 75-1359
Bailey, J.B., 75-0823
Bailie, M.D., 75-1361
Balistreri, W.F., 75-1323
Ballschmiter, K., 75-1217, 75-1220
Baluja, G., 75-1212
Bami, H.L., 75-1475
Banay-Schwartz, M., 75-0882
Bankowska, J., 75-1418, 75-1420
75-1422, 75-1425

Barbier, F., 75-1060
Bardos, T.J., 75-1114
Barkely, J.H., 75-0971
Barnes, A., Jr., 75-1043
Barnes, J.M., 75-0835
Barnett, F.L., 75-1355

Barretto, H.H.C., 75-1291, 75-1292

Batora, V., 75-0989, 75-0990, 75-1248
Bauer, K., 75-0763
Baur, J.R., 75-0761
Beasom, S.L., 75-1309
Beattie, K.L., 75-1180, 75-1396
Bedford, C.T., 75-0880
Beland, F.A., 75-1293
Bell, T.G., 75-1081
Bellin, J.S., 75-1327
Bemelmans, J.M.H., 75-0779
Benke, G.M., 75-0863
Benson, W.W., 75-1387
Benya, T.J., 75-0912
Berencsi, G., 75-0800
Bergel, F., 75-1407
Bergot, B.J., 75-1274
Berkvens, J.M., 75-1444
Berlin, M., 75-1358
Berndt, W.O., 75-1083
Bernsohn, J., 75-1068
Bescol-Liversac, J., 75-1319
Beskid, M., 75-1424
Beskid, W., 75-1421
Bhaskaram, C., 75-1102
Bick, M., 75-1201
Bieniek, D., 75-0879
Biggar, J.W., 75-1011
Binoux, M., 75-1160
Biston, R., 75-0764
Bizeau, E.G., 75-1387
Bjerk, J.E., 75-1005
Blaustein, B.D., 75-0825
Bock, R., 75-0931
Boehme, C., 75-1105
Boghianu, L., 75-0890
Bohme, C., 75-1106
Bojanowska, A., 75-1418, 75-1420
75-1422, 75-1425
Bojovic, B., 75-1272
Bolewski, K., 75-1461
Bollag, J.M., 75-0894, 75-1100, 75-1140
Bollen, W.B., 75-0954
Bond, E.J., 75-0783
Bonner, F.L., 75-1209
Booth, G.M., 75-1277
Booth, N.H., 75-1271
Borkovec, A.J., 75-0961
Borthwick, P.W., 75-1343
Bosshardt, H.P., 75-0978
Boudreau, A., 75-1474
Bovey, R.W., 75-0761
Bowden, J.A., 75-1087
Bowman, M.C., 75-0771
Bradley, R.L., 75-1334
Brankov, K., 75-1332, 75-1446
Braun, H.E., 75-0782, 75-1009, 75-1040
Brenner, F.J., 75-0937
Brenner, K.S., 75-0992
Brestkin, A.P., 75-0859
Brindley, W.A., 75-1377
Bronisz, H., 75-1288, 75-1289
Brooks, G.T., 75-1181
Brown, C.B., 75-1088
Brown, E.A.B., 75-1076, 75-1077
Brown, M.J., 75-1470
Brown, T.R., 75-1090
Brownson, R.H., 75-1175
Bruce, W.N., 75-0769, 75-1265
Brueggemann, J., 75-0799
Brun, G.L., 75-0975
Bruns, V.F., 75-0781
Brunton, R.B., 75-0918
Bryan, G.T., 75-1098
Bublik, L.I., 75-1232, 75-1234
Buck, W.B., 75-0898
Budhraja, T.R., 75-0791
Bulger, W.H., 75-1094
Bull, C.J., 75-1164
Burkser, G.V., 75-1025
Burns, I.G., 75-0807
Burns, J.E., 75-0762, 75-1270
Burns, K.A., 75-1095
Burrage, R.H., 75-0820
Busch, L., 75-0799
Businelli, M., 75-0759
Butler, G.L., 75-1341
Butler, L.R.P., 75-1001
Buttar, H.S., 75-0947
Cahill, W.P., 75-1030
Calciu, A., 75-1369
Calderbank, A., 75-0997
Campbell, K.I., 75-1085
Campbell, R.D., 75-1122
Camper, N.D., 75-0956, 75-1020
Caratozzolo, C., 75-0785
Carey, A.E., 75-1029
Carlson, G.P., 75-1441
Carson, V.G., 75-1176
Carter, G.E., Jr., 75-0956
Casalis, J., 75-0760
Casida, J.E., 75-0895, 75-0896, 75-0926
75-0977, 75-1192, 75-1428
Castles, T.R., 75-1073
Cau, G., 75-1318
Cavazzini, G., 75-1370
Cedeno-Maldonado, A., 75-0897
Celo, J.S., 75-1203
Cencelj, J., 75-1251
Cenci, P., 75-1370
Cerna, M., 75-1023
Cerra, P.E., 75-1093
Chagnon, A., 75-0881
Chambers, H., 75-1356
Chand, N., 75-0892
Chandler, J.H., Jr., 75-0970
Chang, C.S., 75-0986
Charnetski, W.A., 75-0865
Chauhan, U.P.S., 75-1155
Cheung, M.W., 75-1011
Chian, E.S.K., 75-0769, 75-1265
Chiba, T., 75-0867
Chio, L.C., 75-1194
Chiou, C.Y., 75-0858, 75-0969
Chisholm, D., 75-0765
Chiu, C.W., 75-1098
Chmielewski, K., 75-1427
Chmielnicka, J., 75-1405
Choi, P.M.K., 75-0972, 75-1032
Chortyk, O.T., 75-0984
Chow, A.Y.K., 75-1339
Chow, I., 75-1327
Chu, J.P.H., 75-1128

Chugh, K.S., 75-1042
Churchill, L., 75-1176
Cieri, B., 75-1456
Cieslicki, J., 75-1290
Cikrt, M., 75-1000, 75-1132
Civen, M., 75-1088
Clarkson, T.W., 75-0946
Clausen, B., 75-0833, 75-0834
Clerman, R.J., 75-1357
Cliath, M.M., 75-0778
Closset, J.L., 75-0995
Coats, J.R., 75-0910
Cochran, R., 75-1121
Cochrane, T., 75-1121
Cochrane, W.P., 75-1260
Coldwell, B.B., 75-0947
Collins, H.L., 75-1034, 75-1035
Collom, W.D., 75-0821
Colosi-Esca, D., 75-1380
Colvin, H.W., 75-0944
Comba, M.E., 75-0784
Conso, F., 75-1319
Coons, L.B., 75-1347
Copin, A., 75-0995
Coppage, D.L., 75-0950
Coppolo, A., 75-1311
Corbeil, M., 75-0881
Corneliussen, P.E., 75-1033
Corradini, L., 75-1408
Corson, S.D.W., 75-0937
Cortes-Marmolejo, F., 75-1335
Cotman, C.W., 75-1176
Couch, D.B., 75-0941
Cox, J., 75-0988
Cox, J.H., 75-1062, 75-1103
Craig, R.B., 75-1299
Crawford, R.B., 75-1146
Cremlyn, R.J.W., 75-1157
Crisetig, G., 75-1263
Crockett, A.B., 75-1029
Crosby, D.G., 75-0777, 75-1273
75-1276
Crosson, P.R., 75-1240
Cuculis, J.J., 75-1075
Cunningham, D.J., 75-1004
Curtis, R.F., 75-1014, 75-1177
Custovic-Begovic, F., 75-0763
Cwiertniewska, B., 75-1296
Cwiertniewska, E., 75-1294
Czaplicki, E.J., 75-0894
Czeizel, A., 75-1046
Dafter, R., 75-1238
Dalglish, F.W., 75-1176
D'Angelo, A.M., 75-1312
D'Arca Simonetti, A., 75-1287
Darrow, D.C., 75-1279
Darwazeh, H.A., 75-0922
Davidson, J.M., 75-0794
Davies, D.L., 75-1067
Davies, D.S., 75-1067
Davies, R.E., 75-1317
Davis, B.N.K., 75-0776
Davis, J., 75-1034, 75-1035
Davison, K.L., 75-0848, 75-1062
75-1103
Davy, F.B., 75-1208
De Jersey, J., 75-1120
De Matteis, F., 75-1136, 75-1149
De Sloovere, J., 75-1331
De Wolf, L., 75-1056
Deason, T.R., 75-1341
Debackere, M., 75-1331
Debska, W., 75-0803
Dedek, W., 75-1112
Del Vecchio, V., 75-0785
Demint, R.J., 75-0781
Den Tonkelaar, E.M., 75-1443, 75-1444
Dennis, C., 75-1177
Dennison, D.G., 75-1021
Deputat, G.Ye., 75-1267
Desaiah, D., 75-1342
Deshmukh, S.N., 75-1481
Devlin, R.M., 75-0955
Di Blasi, S., 75-1311
Dieter, M.P., 75-1104
Dill, P.A., 75-1165
Dimond, J.B., 75-1259
Dinu, I., 75-0890
Dmitriyeva, O.V., 75-1052
Docekal, O., 75-1127
Dodic, S., 75-0816
Doguchi, M., 75-1258
Doherty, R.A., 75-1324
Doi, E., 75-0899
Domanski, J.J., 75-0986
Dominska, J., 75-1296
Donegan, L., 75-0988
Dooley, K.L., 75-1064, 75-1079
75-1162
Dorer, M., 75-1251
Dorough, H.W., 75-0780, 75-0850
Drescher-Kaden, U., 75-0799
Dube, J.N., 75-1154
Dubey, H.D., 75-1379
Duculot, C., 75-0995
Dudman, N.P.B., 75-1120
Dumas, T., 75-0783
Dunham, L.L., 75-0983, 75-1274
Dure, G., 75-0801
Durrigl, A., 75-1452
Duvenci, J., 75-1457
Dvorchik, B.H., 75-1350
D'yachuk, I.A., 75-1051
Earnest, R., 75-1345
Ebing, W., 75-0993
Ecker, J.L., 75-1070
Eder, G., 75-0980
Edery, H., 75-0860
Edward, G.J., 75-1161
Egle, J.L., Jr., 75-0945
Ehrenberg, L., 75-1395
Eisenbrand, G., 75-1362
El-Bassiouni, E.A., 75-1072
Elbanowska, H., 75-1460
Elder, G.H., 75-1151
Elespuru, R., 75-0923
Eliseyeva, M.A., 75-0994
Elliot, J.M., 75-0872
Elliott, M., 75-1016
England, D.C., 75-0824
Engst, R., 75-0804
Enos, H.F., 75-1215
Epps, E.A., Jr., 75-1257
Erdmann, W.D., 75-1109
Erne, K., 75-0798
Erney, D.R., 75-0973, 75-0974
Ernst, W., 75-0980
Eroschenko, V.P., 75-1439
Ertugrul, G., 75-1255
Estesen, B.J., 75-1030
Eyman, L.D., 75-1183
Fabacher, D.L., 75-1356
Fahmy, M.A., 75-0960
Fang, H.H.P., 75-0769, 75-1265
Farber, T.M., 75-1093
Farmer, J., 75-1065
Farmer, J.H., 75-1079, 75-1162
Farnham, A.W., 75-1016
Farrell, K., 75-1081
Faure, H., 75-1318
Faure, J., 75-1318
Feiner, D., 75-1126
Feller, D.R., 75-1074
Fenster, C.R., 75-1007
Fenwick, R.G., 75-0874
Ferguson, D.M., 75-1264
Fernandez, M.E., 75-1488
Fernandez, O.A., 75-1488
Ferrando, R., 75-0869, 75-0870
Fiedoruk-Poplawska, T., 75-1266
Fields, M., 75-1375
Filipan, T., 75-1250
Fink, G.B., 75-0940
Fischer, G.W., 75-1112
Fishbein, L., 75-1201
Fisher, F.M., 75-1467
Fisher, N.S., 75-0908
Fitak, B., 75-1491, 75-1493
Flashinski, S.J., 75-1123
Fonberg-Broczek, M., 75-1426
Forester, J., 75-1343
Forster, M.S., 75-1152
Fourlon, C., 75-0869, 75-0870
Fowler, J.F., 75-1209
Fox, J.P., 75-1061
Franco, J.M., 75-1010, 75-1212
Frank, P.A., 75-0781
Frank, R., 75-0782, 75-1009, 75-1040
Fraselle, J., 75-0764
Frear, D.S., 75-1195
Freed, V.H., 75-0806
Freitag, K.D., 75-0931
French, J.E., 75-1063
Friedman, M.A., 75-0941
Friend, M., 75-1385, 75-1386
Fries, G.F., 75-1376
Fuchigami, S., 75-1015
Fujimoto, Y., 75-1286, 75-1449
Fukano, S., 75-1258
Fukui, M., 75-1322
Fukui, S., 75-1483
Fukushima, A., 75-1410
Fukuto, T.R., 75-0757, 75-0960
Fuller, G.C., 75-1441
Fusia, E.P., 75-0821
Futenma, M., 75-1305
Gacuhi, D.M., 75-1330
Gaeb, S., 75-1026, 75-1349

Gak, J.C., 75-0869
Galasinska-Pomykol, I., 75-1391
Galea, V., 75-1380
Gamble, W., 75-0854
Gangolli, S.D., 75-1337
Ganz, J.W., 75-1109
Garry, V.F., 75-1091
Gates, A.H., 75-1324
Gaughan, E.J., 75-0958
Gaughan, L.C., 75-0896
Gautier, F., 75-0870
Gazzard, M.F., 75-1139
Gee, J.M., 75-1177
Gee, M.G., 75-0874, 75-1014, 75-1177 75-1223
Gehlbach, S.H., 75-1304 , 75-0811
Gehring, P.J., 75-1361
Gehrs, C.W., 75-1183
Gellert, R.J., 75-0925, 75-1152
Genina, S.A., 75-1054
Georgian, L., 75-1397
Gerakis, P.A., 75-0805
Gergely, A., 75-0882
Gertig, H., 75-1461
Getchell, A.S., 75-1259
Getzin, L.W., 75-1119
Ghachem, A., 75-0830
Ghezzo, F., 75-1408
Ghizelea, L., 75-0890
Gibel, W., 75-0841
Gibson, J.E., 75-1070, 75-1173
Giermaziak, H., 75-1390
Gilbert, M.D., 75-1159
Gilbertson, M., 75-0774
Gill, S.S., 75-0926
Gillette, J.R., 75-1076
Gingell, R., 75-1130
Girard, F., 75-1160
Givran, V., 75-1465
Glasson, B., 75-0936
Glukhova, L.G., 75-1172
Glunz, L.J., 75-0979
Goering, D.K., 75-0768
Goerke, H., 75-0980
Goerttler, K., 75-1111
Golberg, L., 75-1235
Gololobov, V.P., 75-1025
Gonzalez, J., 75-1215
Goodman, G.T., 75-0756
Goodman, N., 75-1246
Gordon, H.L., 75-1303
Gosselin, C., 75-1474
Goto, S., 75-1286
Gourmelen, M., 75-1160
Goursaud, J.G., 75-0760
Graham, C.K., 75-1103
Graillot, C., 75-0869
Grant, D.L., 75-1131
Grant, W.C., Jr., 75-1147
Graves, J.B., 75-1209
Gray, R.A., 75-1428
Greaves, J.H., 75-1167
Greene, F.E., 75-0877, 75-1089 75-1090
Greenhalgh, R., 75-0990, 75-1469
Greichus, Y.A., 75-1038
Greselin, E., 75-0901
Grice, H.C., 75-0948
Griffiths, N.M., 75-0874, 75-1177
Grigor, K.M., 75-0873
Grishchenko, L.I., 75-0953
Grossi, F., 75-0883
Grunow, W., 75-1105, 75-1106
Guarino, A.M., 75-1008, 75-1146 75-1438
Guconic, M., 75-0816
Guertzenstein, P.G., 75-0860
Guglielmini, C., 75-1408
Guildford, S., 75-1279
Guillam, C., 75-1319
Guirguis, G.N., 75-1377
Gunther, F.A., 75-0971
Guthrie, F.E., 75-1193
Gwiazda, J., 75-1493
Gysin, H., 75-0755
Haag, D., 75-1111
Hadzovic, S., 75-1446
Haeberer, A.F., 75-0984
Haegele, M.A., 75-0849
Hagedorn, M.L., 75-0893
Haines, J.R., 75-0846
Halacka, K., 75-1127, 75-1262
Haley, T.J., 75-1064, 75-1079, 75-1162
Hamilton, S.E., 75-1120
Hammock, B.D., 75-0926, 75-0977
Hammock, L.G., 75-0977
Hansen, D.J., 75-1277
Hanstein, W.G., 75-0861
Haque, R., 75-0806
Harbison, R.D., 75-1080
Hario, K., 75-0808
Harmon, J.R., 75-1064, 75-1079 75-1162
Harris, L.W., 75-1091
Harrod, R.K., 75-0880
Hart, G.J., 75-1116
Hartgrove, R.W., 75-1061
Harvey, E.J., Sr., 75-1031
Harvey, G.R., 75-0786
Hassen, M., 75-0830
Hatefi, Y., 75-0861
Hatina, G.V., 75-1131
Hattrup, A., 75-0781
Hayashi, M., 75-0808
Hayes, H.B., 75-0807
Hazelwood, J.C., 75-1082
Heath, D., 75-0917, 75-1059
Heinrichs, W.L.R., 75-0925 , 75-1152
Helling, C.S., 75-1021
Hellmann, B., 75-1230
Hendrickson, C.M., 75-1087
Henis, Y., 75-0935
Henou, C., 75-0871
Henry, N., 75-0870
Henry, P.B., 75-0965
Herbst, M., 75-1442
Herr, F., 75-0901
Herzel, F., 75-1216
Herzlinger, G., 75-0789
Hetzler, H., 75-1037
Heyndrickx, A., 75-1056, 75-1060
Hiesche, K.D., 75-1395
Hill, E.F., 75-1104
Hinojo, J.M., 75-1488
Hirose, M., 75-1410
Hirwe, A.S., 75-1194
Hladka, A., 75-1406
Hodgson, E., 75-1372, 75-1433
Hodgson, J.R., 75-1073
Hodgson, R.A., 75-1337
Hoermann, W.D., 75-1222
Hoffman, R.V., Jr., 75-1334
Holder, B.B., 75-1303
Holdrinet, M., 75-1009
Hook, G.E.R., 75-0856, 75-0857
Hook, J.B., 75-1070, 75-1361
Hoorens, J., 75-1331
Hoppe, P., 75-0799
Horacek, J., 75-1459
Hori, S., 75-0943
Horowitz, M., 75-0789, 75-0934 75-0935
Hoskam, E.G., 75-1219
Hosoda, K., 75-0920
Howes, B.L., 75-0868
Hruska, J., 75-1261
Hsu, J.M.C., 75-1020
Hudgins, P.M., 75-0945
Hudson, R.H., 75-0849
Huguet, M., 75-0822
Hulin, N., 75-0789, 75-0934, 75-0935
Hurter, J., 75-1218
Huschenbeth, E., 75-1249
Hutzinger, O., 75-0972
Hyde, K.M., 75-1209
Iida, K., 75-1015
Iizuka, S., 75-0921
Illingworth, C.M., 75-0837
Impson, J.W., 75-1257
Imre, R.A., 75-0800
Ino, T., 75-0921
Inokuchi, K., 75-1145, 75-1189
Inuyama, Y., 75-1297, 75-1298
Irving, C.C., 75-0948
Isenberg, F.M., 75-1159
Ishide, N., 75-0808
Ishiguro, S., 75-1365, 75-1366
Ishikawa, S., 75-1048, 75-1338
Ishikura, H., 75-1002
Ito, N., 75-1412
Ito, S., 75-0808
Iverson, F., 75-1131
Ivey, M.C., 75-1468
Iwami, T., 75-1145
Iwamoto, T., 75-0920
Iyatomi, K., 75-1400
Izumiyama, T., 75-1057
Jaggi, C.B., 75-1155
Jan, J., 75-0763
Janes, N.F., 75-0787, 75-1016
Jaros, F., 75-1326
Jenkins, D., 75-1003
Johannsen, F.R., 75-1353
Johnson, C.D., 75-1450
Johnson, D.W., 75-1122
Johnson, J.C., Jr., 75-0771

Johnson, J.H., 75-0949
Johnson, J.T., 75-1361
Johnson, M.K., 75-1118
Johnson, W.H., 75-0986
Johnson, W.W., 75-0887
Jolley, R.J., 75-1183
Jones, R.L., 75-0771
Joubert, S.M., 75-1137, 75-1150
Jurek, A., 75-1200, 75-1494
Kacew, S., 75-0853
Kadyrov, U.Z., 75-1170
Kaise, T., 75-1015
Kaiser, T.E., 75-1300
Kalic Filipovic, D., 75-0816
Kaloyanova, F., 75-1050
Kamata, K., 75-1057
Kamata, T., 75-0808, 75-1268
Kamau, J.A., 75-1330
Kamilov, I.K., 75-1169
Kanayev, A.I., 75-0953
Kanazawa, J., 75-1019
Kanbayashi, Y., 75-0920
Kaneko, Y., 75-0942
Kapor, S., 75-1363
Kar, P.P., 75-0932
Karczmar, A.G., 75-1068
Karczmarczyk, S.J., 75-0955
Karlog, O., 75-0833, 75-0834
Karnaky, K.J., 75-1086
Kasakawa, H., 75-1189
Kasang, G., 75-0914
Kasatiya, S.S., 75-0881
Kashimoto, T., 75-0943
Kasper, P., 75-1392
Kataoka, S., 75-1186, 75-1187
Kato, T., 75-1058
Kaufman, D.D., 75-1021
Kavanagh, W.G., 75-1075
Kawahara, T., 75-1286
Kawai, M., 75-1190
Kawajiri, M., 75-1388
Kawamura, A., 75-0942
Kawatski, J.A., 75-1351
Kay, J.M., 75-0917
Kay, K., 75-1307, 75-1445
Kazachuk, Yu.S., 75-1171
Kazantzis, G., 75-0836
Kenaga, E.E., 75-0964
Kendrick, J.B., Jr., 75-1236
Kennedy, G.L., Jr., 75-1174
Keplinger, M.L., 75-1174
Kern, A.D., 75-0903
Kettmann, R., 75-0995
Khalifa, S., 75-0895
Khan, H.M., 75-0847, 75-1301
Khan, M.A.Q., 75-0847, 75-1301 75-1340, 75-1431
Khan, S.U., 75-0802, 75-1006, 75-1466
Kharchenko, S.M., 75-1393
Khasawinah, A.M., 75-0962
Khmel'nitskaya, V.N., 75-0994
Khokhol'kova, G.A., 75-1144
Kilzer, L., 75-0879
Kimball, R.F., 75-1396
Kimmel, E.C., 75-1192
Kimmerle, G., 75-1108
Kimura, M., 75-1145, 75-1189
Kino, A., 75-0842
Kinter, W.B., 75-1066, 75-1086 75-1097, 75-1148
Kirkwood, J.I., 75-1206
Kiseleva, N.I., 75-1233
Klaas, E.E., 75-1300
Kleerekoper, H., 75-1208
Klein, W., 75-0879
Klimmer, O.R., 75-1108
Klingebiel, U.I., 75-1012
Klisenko, M.A., 75-1282
Klobucar, M., 75-1451
Klos, H., 75-1421, 75-1424
Knauf, W., 75-1389
Knight, L.A., Jr., 75-1031
Knowles, C.O., 75-1353, 75-1430
Knox, F.E., 75-0771
Knox, J.R., 75-0895
Kobayashi, S., 75-1057
Koch, R.B., 75-1342
Koellmer, H., 75-1442
Koivisto, O., 75-1302
Kolesnikov, Yu.G., 75-0952
Kolmodin-Hedman, B., 75-0815
Kolosovskaya, V.M., 75-1448
Kolycheva, S.S., 75-1207
Konetzke, G.W., 75-0813
Konishi, Y., 75-1412
Kono, I., 75-1365
Konstantinova, A.V., 75-1025
Kontek, M., 75-1290, 75-1321, 75-1325
Kopec, J., 75-1333
Koransky, W., 75-0924
Koren, E., 75-0790
Korn, S., 75-1345
Korte, F., 75-0879, 75-1026, 75-1349
Koschier, F., 75-1083
Koshakji, R.J., 75-1080
Kosmatyy, Ye.S., 75-1232, 75-1234
Kotangle, R.S., 75-1475
Kovac, J., 75-1478
Kovacicova, J., 75-1248, 75-1469
Kovalcik, V., 75-1346
Kowianska, E., 75-1296
Kozak, H., 75-1285
Kozlowski, B., 75-1231
Kralik, P., 75-1478
Krampl, V., 75-1406
Krasil'shchikov, D.G., 75-1018
Kratinova, R., 75-1326
Krauthacker, B., 75-1117
Kravets, Ye.V., 75-1027
Krempler, F., 75-1254
Kroes, R., 75-1443, 75-1444
Krupka, R.M., 75-0929
Kubacki, S.J., 75-1290
Kuehl, D.W., 75-0982
Kuga, T., 75-1191
Kulkarni, A.P., 75-1372
Kupfer, D., 75-1094
Kuroi, N., 75-1015
Kuwabara, N., 75-1411
Kuwahara, Y., 75-1283
Kvalvag, J., 75-0996
Kynard, B., 75-1210
Kyurkchiyev, S., 75-1055
La Rocca, P.T., 75-1441
Lahaniatis, E.S., 75-1349
Lajtha, A., 75-0882
Lake, B.G., 75-1337
Lakshminarayana, V., 75-1482
Lalka, D., 75-1114
Land, D.G., 75-1014, 75-1177, 75-1223
Lande, S.S., 75-1245
Landgraf, H., 75-0924
Lang, S., 75-1099
Langone, J.J., 75-1229, 75-1487
Lara, W.H., 75-1291, 75-1292
Lareng, L., 75-1344
Larose, R.H., 75-1476
Larsen, J.R., 75-1277
Laver, G.W., 75-1479
Lavy, T.L., 75-0991, 75-1007
Lawley, P.D., 75-0875
Lawrence, J.F., 75-0976, 75-1473 75-1479
Le Patourel, G.N.J., 75-1184
Leadem, T.P., 75-1122
Leberl, C., 75-0924
Lech, J.J., 75-0951
Lee, C.C., 75-1073
Lee, H.M., 75-1084
Lee, K.C., 75-1355
Lee, O.S., 75-1074
Lee, Y.W., 75-0820
Leffingwell, J.T., 75-1003
Lenz, D.E., 75-1075
Leoni, V., 75-0785
Leuck, D.B., 75-0771
Levac, L.D., 75-0913
Lewin, J.F., 75-1044
Lewinsky, B.S., 75-0873
Lewis, D.L., 75-1134
Lewis, T.D., 75-0829
Liang, G.H., 75-1355
Lichtenstein, E.P., 75-1123
Lijinsky, W., 75-0923
Lin, T., 75-0850
Lin, T.H., 75-1373, 75-1374
Lincer, J.L., 75-0772
Linder, R.L., 75-1038
Lindgren, J.E., 75-1107
Lingens, F., 75-1359
Linko, R.R., 75-0775
Lisk, D.J., 75-1159
Lister, N.A., 75-1032
Liu, K.C., 75-1140
Liu, L.C., 75-0897
Lloyd, A.G., 75-1337
Locke, L.N., 75-1113
Lockyer, N.F., 75-0918
Lokke, H., 75-0788
Long, P.P., 75-0915
Longland, R.C., 75-1337
Loos, M.A., 75-1124
Lopez, C.E., 75-1206
Losito, L., 75-1312
Love, J.L., 75-1044
Lu, P.Y., 75-1194
Lucier, G.W., 75-0856, 75-0857 75-1071

Lucuta, V., 75-1380
Luczak, J., 75-1199
Ludke, J.L., 75-1104
Ludwicki, J.K., 75-1414
Lui, H., 75-1360
Lundy, H.W., 75-1278
Luquet, F., 75-0760
Lutomski, J., 75-0803
Luton, J.P., 75-1160
Lutz-Ostertag, Y., 75-0871
Lyubenko, P.Kh., 75-1281
Macdonald, S.R., 75-0985
Machin, A.F., 75-0787
MacRae, I.C., 75-1203
Maeda, H., 75-1286
Magee, P.S., 75-0957
Magos, L., 75-0946
Mahoney, J.J., Jr., 75-0866
Mailman, R.B., 75-1372
Maitai, C.K., 75-1330
Makarova, S.V., 75-0994
Makhija, S.J., 75-1348
Maki, A.W., 75-0906
Makino, F., 75-1115
Makiura, S., 75-1412
Malby, M., 75-0822
Maleszewska, J., 75-1199
Maling, H.M., 75-1076, 75-1077
Mallet, V., 75-0975, 75-1221
Malygina, Ye.F., 75-1464
Mandell, B., 75-1099
Manikowski, W., 75-1320
Manko, Yu.P., 75-1393
Mann, H.D., 75-1468
Mann, J.B., 75-1215
Manske, D.D., 75-1033
Marcelja, L., 75-1451, 75-1452
Marchenko, P.V., 75-1027
Marcinkowska, B., 75-1325
Marcinkowski, T., 75-1320
Maren, T.H., 75-1350
Marka, C., 75-1318
Markiewicz, K., 75-1405
Markin, G.P., 75-1034, 75-1035 75-1036
Markkula, M., 75-0826
Markova, V., 75-1478
Marks, C.F., 75-0872
Marks, R.M., 75-1093
Marrow, G.S., 75-1376
Marshall, C.D., 75-0938
Marshall, W.D., 75-0990
Martens, P.H., 75-0764, 75-0995
Martin, G.B., 75-1474
Marucchini, C., 75-0759
Mason, M., 75-1085
Matin, M.A., 75-0932, 75-1138
Matokhnyuk, L.A., 75-1144
Matsumoto, K., 75-1188
Matsumura, F., 75-1429
Matsumura, T., 75-1284
Matsunaga, A.K., 75-1454, 75-1455
Matsuoka, M., 75-1458
Matsushima, S., 75-1178
Matthews, E., 75-0950
Matthews, H.B., 75-1375
Matthysse, J.G., 75-0998, 75-1243
Matyushina, V.I., 75-1144
McBain, J.B., 75-0959
McCaughey, H., 75-1314
McClure, G.W., 75-1028
McConnell, G., 75-1264
McDaniel, O.S., 75-0856
McDonald, M.J., 75-1351
McDowell, J.R., 75-1271
McEwen, F.L., 75-0782, 75-1040
McInerney, J.E., 75-1164
McKinney, J.D., 75-1072
McLeod, H.A., 75-0976
McQueen, A., 75-1121
McWade, J.W., 75-1009
Meehan, W.R., 75-1383
Mehendale, H.M., 75-1072, 75-1375
Meith-Avcin, N., 75-1166
Mendoza, C.E., 75-1480
Menezes, A.P., 75-1041
Menn, J.J., 75-0958, 75-0959
Menzer, R.E., 75-0927, 75-1373 75-1374
Mercier, M., 75-1107
Messick, J.P., 75-1387
Metcalf, R.L., 75-1194
Meyer, H.C., 75-1075
Michalek, M., 75-0989
Mick, D.L., 75-1037
Mihara, K., 75-0852
Millemann, R.E., 75-1382
Miller, D.S., 75-1066, 75-1097
Miller, F.M., 75-0762, 75-1270
Miller, P.E., 75-0940
Miller, R.W., 75-0915
Miller, W.W., 75-0983
Minakami, S., 75-0900
Minard, R.D., 75-0894
Mitchell, R., 75-1182
Mitchell, W.G., 75-1029
Miura, K., 75-0921
Miura, T., 75-1226
Miyachi, Y., 75-1214
Miyamoto, J., 75-0852, 75-0928
Miyao, N., 75-1365, 75-1366
Miyata, T., 75-1400
Mizuhira, V., 75-1381
Moilanen, K.W., 75-1276
Moitra, B., 75-1475
Monaco, T.J., 75-0795
Monaghan, C.F., 75-1032
Montgomery, M.L., 75-1256
Moore, J.A., 75-0857, 75-1437
Mora, A., 75-1263
Moreale, A., 75-1024
Morici, G., 75-1311
Morita, H., 75-0773, 75-1280
Moriyama, I., 75-1202
Moroi, K., 75-1191
Morse, C.S., 75-1462
Mosser, J.L., 75-0864
Mueller, K., 75-0992
Mulla, M.S., 75-0922
Mullins, W.H., 75-1387
Munch, B., 75-0833
Mundy, D.E., 75-0787
Munnecke, D.M., 75-0907
Murado, M.A., 75-1212
Murai, S., 75-0920
Murata, M., 75-1398
Murphy, S.D., 75-0863, 75-1339
Murrill, E., 75-1073
Murzabekov, Sh.M., 75-1207
Musac, I., 75-1250
Mussell, D.R., 75-0963
Mussman, H.C., 75-0770
Musty, P.R., 75-1477
Muzrabekov, Sh.M., 75-1169
Nadzhimutdinov, K.N., 75-1169 75-1207
Nagano, H., 75-1015
Nagasaki, H., 75-1412
Nagata, H., 75-1057
Nagler, M., 75-1394
Nagy, J.G., 75-0919
Naito, M., 75-1190
Nakae, C., 75-0928
Nakagawa, M., 75-0777, 75-1101 75-1273
Nakamura, A., 75-0900
Nakamura, T., 75-1101
Nakazawa, N., 75-1115
Namdeo, K.N., 75-1154
Narayanaswami, K., 75-1475
Nazer, I.K., 75-0905
N'Diaye, A.L., 75-0870
Nebert, D.W., 75-1179
Needham, P.H., 75-1016
Neskovic, N., 75-1363
Neville, A.M., 75-0873
Neville, G.A., 75-1358
Newsome, W.H., 75-1471
Nicholson, S.A., 75-1357
Nickless, G., 75-1477
Nikonorow, M., 75-1414
Ninfo, V., 75-1408
Nishikawa, M., 75-1284
Nishikawa, T., 75-1058
Nishimura, M., 75-0942
Nishio, Y., 75-1409
Nitsche, I., 75-1220
Njoroge, S., 75-1330
Noda, K., 75-0867
Norris, L.A., 75-0954, 75-1256, 75-1383
North, H.H., 75-1373, 75-1374
Norval, E., 75-1001
Nowaczyk, W., 75-1461
Nukiyama, M., 75-1015
Nys, L., 75-0764
Oberholser, K.M., 75-1089
O'Brien, R.D., 75-1116
Obuchowska, I., 75-1295
Ochynski, J., 75-1288, 75-1289
Ogata, S., 75-0942
Ogawa, E., 75-1388
Ohkawa, H., 75-1192
Ohkawa, R., 75-1192
Ohlendorf, H.M., 75-1300
Ohsawa, T., 75-0895
Okada, S., 75-1115
O'Kelley, J.C., 75-1341
Oliver, R.M., 75-1306

Olson, R.J., 75-0854
Onuska, F.I., 75-0784
Orr, D.J., 75-0875
Orton, T.C., 75-0857
Oshima, Y., 75-0773, 75-1280
Osterman-Golkar, S., 75-1395
Otaci, C., 75-1255
Ott, M.G., 75-1303
Ouw, K.H., 75-1316
Overstreet, D.H., 75-1176
Owen, R.B., Jr., 75-1259
Owens, I.S., 75-1179
Ozeranschi, L., 75-0890
Padgett, G., 75-1081
Padmanaban, G., 75-1153
Palut, D., 75-1423
Palva, H.L.A., 75-1302
Palva, I.P., 75-1302
Panno, G., 75-1311
Pant, N.C., 75-1354
Panzel, H., 75-1217
Paradowski, S., 75-1290, 75-1321
Paris, D.F., 75-1134
Parkhomenko, T.V., 75-0859
Parlar, H., 75-1026, 75-1349
Parr, L.J., 75-1014
Partin, J.C., 75-1323
Passanati, G.T., 75-1089
Patil, T.N., 75-0891
Patterson, R., 75-1384
Paul, C.J., 75-0947
Paulov, S., 75-0843
Pawar, S.S., 75-1348
Pazderova, J., 75-1000
Peakall, D.B., 75-1066, 75-1097 75-1148
Pearson, C.R., 75-1264
Peel, J.L., 75-1177
Pekas, J.C., 75-1440
Pendergrass, A., 75-1159
Peoples, A., 75-1162
Peoples, S.A., 75-1078
Perera, M.V.F., 75-1045
Perlovskaya, E.D., 75-1171
Perna, A., 75-1456
Pernov, K., 75-1055
Perry, A.S., 75-1436
Perry, J.A., 75-0979
Pesic, V., 75-1446
Peterson, R.H., 75-1163
Petrasek, R., 75-1127
Petre, I., 75-1369
Petrella, V.J., 75-1061
Pevzner, D.L., 75-1211
Pflugmacher, J., 75-0993
Pham-Huu-Chanh, 75-1344
Phillips, R.W., 75-1082
Picer, M., 75-1252, 75-1253
Picer, N., 75-1252, 75-1253
Pieczko-Kuduk, I., 75-1403
Piedade, J.R., 75-1453
Pietraszek, Z., 75-1290, 75-1321 75-1325
Pilichowska, J., 75-1404
Pilinskaya, M.A., 75-1049
Pimentel, D., 75-1246
Pimentel, J.C., 75-1041
Pinegar, J.A., 75-0988
Pis'mennaya, M.V., 75-1282
Plapp, F.W., Jr., 75-1434
Plestina, R., 75-1364
Plimmer, J.R., 75-1012
Poirier, L.A., 75-0948
Polen, P.B., 75-0987
Polin, D., 75-1125
Popendorf, W.J., 75-0810
Popova, M., 75-1463
Poser, W., 75-1109
Preiss, D., 75-1111
Preissig, S.H., 75-1096
Preussmann, R., 75-1362
Pringle, J.D., Jr., 75-0781
Pritchard, J.B., 75-1069, 75-1086 75-1148, 75-1438
Prodanovic, M., 75-0816
Prouty, R.M., 75-1113
Prozorovskiy, V.B., 75-1168
Pruzandky, J.J., 75-1384
Przybylski, Z., 75-1244
Puccetti, G., 75-0785
Pugh, G.J.F., 75-1205
Pulman, D.A., 75-1016
Purkayastha, R., 75-1466
Quentin, K.E., 75-0801
Quick, M.P., 75-0787
Quistad, G.B., 75-1158, 75-1275
Rabsztyn, T., 75-1333
Radeleff, R.D., 75-0949
Radhakrishnamurty, R., 75-0891
Radomanski, T., 75-1022
Ragab, M.T.H., 75-0766, 75-1039
Rajamanickam, C., 75-1153
Rajapakse, C.N.A., 75-1045
Rall, D.P., 75-1438
Ramachandran, M., 75-1161
Ramachandran, S., 75-1045
Ramsteiner, K.A., 75-1222
Ranke-Rybicka, B., 75-1413, 75-1416
Rantimaki, P., 75-0775
Rao, G.H., 75-0855
Rao, G.M., 75-1355
Rastogi, V.K., 75-1155
Raux, M.C., 75-1160
Rawls, R.L., 75-1237
Reddy, G., 75-1340, 75-1431
Redfern, R., 75-1167
Regan, A., 75-1254
Rehn, M., 75-0818, 75-0827
Reichel, W.L., 75-1113
Reif, V.D., 75-1129
Reiner, E., 75-1117
Reinert, R.E., 75-0916
Ricciardi, A.J., 75-1489
Rickett, F.E., 75-0965
Riera, A., 75-1379
Ringer, R.K., 75-1125
Rip, J.W., 75-0904
Risebrough, R.W., 75-1066, 75-1148
Ritcey, G., 75-0782
Robinson, D., 75-1014, 75-1177 75-1223
Rodionov, G.A., 75-1336
Rodman, J.S., 75-1099
Rodriguez, R.L., 75-1379
Rogers, R.L., 75-0902
Rose, M.S., 75-0862
Rosenthal, J., 75-0982
Rozenfel'd, A.A., 75-1281
Rozengart, V.I., 75-1211
Rudd, R.L., 75-1299
Rump, S., 75-1333
Russell, J.B., 75-1260
Russell, R.L., 75-1450
Russell, R.W., 75-1176
Russev, P., 75-1463
Rutschky, C.W., III, 75-0938
Ruzieska, P., 75-1046
Rybak, M., 75-1419
Sabo, D., 75-1095
Sadasivan, G., 75-1102
Saha, J.G., 75-0820
Saha, M., 75-0820
Saint Ruf, G., 75-1344
Saito, T., 75-1400
Sakakibara, J., 75-1214
Sakama, H., 75-0942
Sakamoto, Y., 75-1269
Samaha, R.J., 75-1043
Sameoto, D.D., 75-1279
Samosh, L.V., 75-0839
Sand, P.F., 75-1029
Sanders, H.O., 75-0970
Sano, I., 75-1308
Santelmann, P.W., 75-0794
Sarkar, B.C.R., 75-1155
Sarna, L.P., 75-0985
Satio, Y., 75-1015
Sato, N., 75-1409
Sato, T., 75-1191
Sato, Y., 75-1483
Sattel, P., 75-0992
Satyanarayana, T., 75-1119
Sauer, H.H., 75-0978
Saul, W., 75-1076
Saunders, R.C., 75-1165
Savic, S., 75-0816
Saxena, J., 75-0878
Scarponi, L., 75-0759
Schaefer, R.G., 75-0980
Schafer, E.W., Jr., 75-0918
Schafer, W., 75-1359
Schein, L.G., 75-1133
Scheufler, H., 75-1112
Schmid, A., 75-0797
Schnaak, W., 75-0804
Schneeweis, J.C., 75-1038
Schooley, D.A., 75-1158, 75-1274 75-1275
Schubert, W.K., 75-1323
Schulte-Hermann, R., 75-0884, 75-0924
Schulze, E.F., 75-1389
Schwartz, C.C., 75-0919
Schwartzel, E.H., 75-0878
Scott, R.D.M., 75-1197
Sears, H.S., 75-1383
Seawright, A.A., 75-1198
Seguchi, K., 75-1225
Seiber, J.N., 75-1276

Sekiguchi, F., 75-1280
Selenka, F., 75-1220
Sell, J.L., 75-0848
Semenova, L.N., 75-1484
Serat, W.F., 75-0823
Serov, V.M., 75-1025
Setlow, J.K., 75-0923
Severn, B.J., 75-1337
Sficas, A.G., 75-0805
Shafik, T.M., 75-1213
Shah, S.A., 75-0875
Shandar, A.G., 75-1316
Shanley, B.C., 75-1137, 75-1150
Sharma, B.K., 75-1042
Shchitskova, A.P., 75-1368
Sheets, T.J., 75-0795, 75-0986
Sherburne, J.A., 75-0772
Sherma, J., 75-1213
Shevchenko, M.A., 75-1027
Shidno, N., 75-1242
Shlevin, E., 75-0790
Shoemaker, J.P., 75-1334
Shotwell, T.K., 75-0758
Shoyama, M., 75-1214
Siddall, J.B., 75-1274
Sidell, R.R., 75-0939
Sides, P.J., Jr., 75-0845
Sieber, S.M., 75-1432
Sigrist, R., 75-1218
Sikka, H.C., 75-0878
Simeon, V., 75-1117
Simeone, A.M., 75-0785
Simpson, G.R., 75-1315
Singh, G., 75-1141, 75-1354
Singh, J., 75-1481
Singh, K., 75-0791
Singhal, P.C., 75-1042
Singhal, R.L., 75-0853
Sinsheimer, J.E., 75-1129
Sjerobabski, D., 75-1250
Skalsky, H.L., 75-1193
Skrinjaric-Spoljar, M., 75-1117
Skroch, W.A., 75-0795
Skromne-Kadlubik, G., 75-1335
Slach, E., 75-1037
Slukan, V., 75-1451
Smilie, J.L., 75-1257
Smirnov, V.S., 75-0952
Smith, E.H., 75-1009
Smith, L.L., 75-0862
Smith, M.S., 75-0930
Smith, P., 75-0917, 75-1059
Smith, R.J., Jr., 75-0793
Smolinska, E., 75-1405
Sniezynski, C., 75-1290
Soderquist, C.J., 75-1276
Sokic, B., 75-1272
Sone, K., 75-1145
Sosnierz, M., 75-1185
Spear, R.C., 75-0810, 75-1003
Spence, J.H., 75-1036
Spencer, W.F., 75-0778
Spirydonow, E., 75-1491
Srinivasan, R., 75-1068
Stahl, P., 75-1099
Staiger, L.E., 75-1158, 75-1275
Stanislawska, J., 75-1199, 75-1413
Stanton, R.H., 75-1431
Starek, A., 75-1367
Starr, R.I., 75-1004
Statham, C.N., 75-0951
Stec, J., 75-1022
Stefanska-Sulik, E., 75-1391, 75-1403
Stefanskiy, K.S., 75-1281
Stern, P., 75-0851
Sternberger, L.A., 75-1075
Stevens, J.T., 75-0877, 75-0933
75-1092
Stevens, W., 75-1060
Stevens, W.E., 75-0865
Stevenson, J.H., 75-1016
Stich, H.F., 75-0948
Stieglitz, R., 75-0812, 75-0841
Stitcher, D.L., 75-1091
Stobbe, H., 75-0812, 75-0841
Stokes, S., 75-1209
Stoltz, D.R., 75-0948
Stone, L.J., 75-0916
Stoops, J.K., 75-1120
Stowers, K.L., 75-0954
Strang, R.H., 75-0902
Street, J.C., 75-1371
Strik, J.J.T.W.A., 75-0886, 75-1135
Strohal, P., 75-1252, 75-1253
Strother, A., 75-0885
Strzelec, A., 75-1196
Stuart, E., 75-0814
Stuneyeva, G.I., 75-1142
Sugano, S., 75-1483
Sugata, Y., 75-0946
Sugaya, H., 75-0808, 75-0809
Summer, A.K., 75-0820
Sundell, L., 75-0818, 75-0827
Surette, D.P., 75-1221
Sustek, J., 75-0989
Sutherland, D.J., 75-1301
Suwala, Z., 75-1421, 75-1424
Suzuki, H., 75-1057, 75-1338
Suzuki, M., 75-1472
Suzuki, S., 75-1388
Suzuki, T., 75-0928, 75-1378
Svetlicic, B., 75-0968, 75-1364
Swanson, H.R., 75-1195
Swartz, R.D., 75-0939
Sweeney, G.D., 75-1360
Swerdloff, R., 75-0925
Symington, T., 75-0873
Syrowatka, T., 75-1200, 75-1494
Szabo, I., 75-1046
Szczurek, Z., 75-1185
Szyszkowska, A., 75-1421, 75-1424

Tafuri, F., 75-0759
Tagatz, M.E., 75-1343
Tai, H., 75-1029
Takagi, F., 75-1015
Takahashi, K., 75-0808
Takahashi, M., 75-0808, 75-1145
75-1189, 75-1410
Takahashi, R., 75-0808
Takahashi, S., 75-0808
Takano, S., 75-1057
Takaori, S., 75-1399
Takashita, T., 75-1297
Takayama, S., 75-1411
Takeda, A., 75-1228
Takeda, S., 75-1399
Taljaard, J.J.F., 75-1137, 75-1150
Talley, D.S., 75-1047
Tanaka, A., 75-1449
Tappan, W.B., 75-1278
Taran, P.N., 75-1027
Tarmas, J., 75-1403
Tarsitani, G., 75-1287
Tatematsu, M., 75-1410
Teller, D.N., 75-0882
Temperli, A., 75-1218
Ten Noever de Brauw, M.C., 75-0779
Teng, T.C., 75-0864
Terziev, G., 75-1463
Teshima, S.I., 75-0842
Thielemann, H., 75-0981
Thier, H.P., 75-0967
Thimann, K.V., 75-0792
Thomas, B.H., 75-0947
Thomas, D.P., 75-1139
Thomas, J.A., 75-1133
Thompson, J.E., 75-1183
Thompson, J.F., 75-1215
Thomson, J., 75-1121
Tibenska, M., 75-1459
Tichy, M., 75-1132
Tiedje, J.M., 75-0893
Tilles, H., 75-1428
Tinworth, H., 75-1167
Tjaelve, H., 75-1110
Tolkmith, H., 75-0963
Tomita, K., 75-1398
Tomita, M., 75-0942
Totaro, S., 75-1311
Train, R.E., 75-1241
Trainer, D.O., 75-1385, 75-1386
Trebicka-Kwiatkowska, B., 75-1022
Trondina, G.A., 75-0953
Truhaut, R., 75-0869, 75-1344
Trumble, T.E., 75-0854
Tsendrovskaya, V.A., 75-1464
Tseng, Y.C.L., 75-0927
Tsuda, H., 75-1410
Tsukahara, I., 75-1399
Tsutsui, J., 75-1058
Tsuzuki, H., 75-1388
Tu, C.M., 75-0872
Tuglular, R., 75-1255
Turhan, K., 75-1255
Turner, C.R., 75-0966
Ubaydullayev, R.U., 75-1143
Uchiyama, M., 75-0867, 75-1378
Ueda, K., 75-0896, 75-0942
Uemura, K., 75-1409
Uhler, R., 75-1064
Ulasevich, E.I., 75-1393
Une, F., 75-0773
Ungerer, O., 75-1362
Urpo, K., 75-0775
Ushiyama, S., 75-1191
Usuya, S., 75-1017
Van Bao, T., 75-1046

Van Beek, H., 75-1219
Van Bladel, R., 75-1024
Van Esch, G.J., 75-1444
Van Faassen, H.G., 75-1204
Van Haverbeck, G., 75-1344
Van Hoof, F., 75-1056
Van Noort, H.R., 75-1329
Van Osten, G.K., 75-1173
Van Peteghem, C., 75-1056, 75-1060
Van Vunakis, H., 75-1229, 75-1487
Vardell, H.H., 75-1013
Vargova, M., 75-1346
Vasques, R.M.P., 75-1454, 75-1455
Vasquez, B.J., 75-1176
Veith, G.D., 75-0982
Vela-Muzquiz, R., 75-1392
Verschuuren, H.G., 75-1443, 75-1444
Vettorazzi, G., 75-1247
Vidakovic, A., 75-0816
Vidal, P.A., 75-1489
Vijayan, V.K., 75-1175
Villeneuve, D.C., 75-1131
Virgo, B.B., 75-0909
Visacki, V., 75-1239
Visek, W.J., 75-1084
Vitorovic, S., 75-1363
Viviani, R., 75-1263
Vojvodic, V.B., 75-0817
Volokhvyanskiy, Ye.A., 75-1170
Vos, J.G., 75-1437
Vrochinskiy, K.K., 75-1053, 75-1171
Vyboh, P., 75-0989
Vymetal, F., 75-1262
Wada, H., 75-1015
Waggoner, T.B., 75-0962
Wagner, S.R., 75-1089
Wagstaff, D.J., 75-1082
Wainwright, M., 75-1205
Wakai, S., 75-1145, 75-1189
Wakasugi, C., 75-1322
Wakatsuki, S., 75-1328
Walsh, A.H., 75-0911
Walsh, D.F., 75-0970
Walsh, F., 75-1182
Walther, W.C., 75-0864
Wang, C.Y., 75-1098
Wang, W.L., 75-0944
Ward, D.V., 75-0868
Ware, G.W., 75-1030
Watanabe, K., 75-1015
Watanabe, T., 75-1286, 75-1472
Wazna-Bogunska, C., 75-1185
Webb, R.E., 75-1061
Webster, G.R.B., 75-0985
Weeraratna, S., 75-0930
Weil, B.Q., 75-0825
Weil, L., 75-0801
Weil, T.A., 75-0825
Weisbart, M., 75-1126
Weisburger, J.H., 75-0948
Weiss, N.N., 75-0914
Weisse, I., 75-1442
Weissglas, G., 75-0819
Weller, T.H., 75-0754
Wennberg, I., 75-1395
Werner, W., 75-0841
Westberg, G.L., 75-1078
Westhoff, D.D., 75-1043
Westlake, W.E., 75-0971
Wheeler, L., 75-0885
Wheeler, W.B., 75-1278
White, J.G., 75-0855
Wiersma, G.B., 75-1029
Wildish, J., 75-1032
Wilhelm, K., 75-0968, 75-1364
Wilkins, J.S., 75-0983
Williford, W.A., 75-0916
Williams, G.M., 75-0876
Williams, M.A., 75-1076
Williams, W.A., 75-0811, 75-1304
Willis, D.E., 75-1352
Wilson, W.O., 75-1439
Winek, C.L., 75-0821
Witkowska, I., 75-1285
Wittels, B., 75-0845
Witzel, D.A., 75-0949
Wojcik, J., 75-1415, 75-1421, 75-1424
Wolfe, N.L., 75-1134
Wolkoff, A.W., 75-1476
Wong, L., 75-1467
Woodin, A.M., 75-0888, 75-0889
Woodrow, J.E., 75-1276
Wright, A., 75-0862
Wright, D.J., 75-1184
Wu, C.H., 75-0794
Wurster, C.F., 75-0864
Wustner, D.A., 75-0960
Wyatt, I., 75-0862
Yacoub, M., 75-1318
Yakawa, K., 75-1409
Yakiich, R.W., 75-0955
Yakubovsky, S.V., 75-1393
Yamada, A., 75-1313
Yamada, T., 75-1269
Yamaguchi, M., 75-0999
Yamane, S., 75-0842
Yamato, Y., 75-1472
Yamawake, T., 75-1015
Yarbrough, J.D., 75-1347, 75-1435
Yasuda, N., 75-1365, 75-1366
Yomeya, N., 75-0942
Yoneda, H., 75-1454, 75-1455
Yoshida, K., 75-1284
Yoshioka, Y., 75-1401, 75-1402
Yoshizaki, H., 75-1485, 75-1486
Younger, R.L., 75-0949
Yu, C.C., 75-1277
Zaborowska, W., 75-1285
Zadrozinska, J., 75-1417, 75-1490
Zakirov, U.B., 75-1170
Zarkovic, G., 75-0763
Zawadzka, H., 75-1460
Zelinskaya, O.V., 75-1143
Zemaitis, M.A., 75-0877
Zerner, B., 75-1120
Zero, M., 75-1492
Zezelj, A., 75-1363
Zhumamudinov, S., 75-1025
Zieba, B., 75-1491
Zielhuis, R.L., 75-1050
Zinkl, J.G., 75-1437
Zitko, V., 75-0972, 75-1032
Zweig, G., 75-0878
Zycinski, D., 75-1199

Corporate Author Index

FAO-WHO, 75-0796
Agric Chem Inspect Stn, 75-1227
Div Protect Living Environ, 75-1310
Who Sci Gr Chem Biochem Method P-estic, 75-1447

The Administrator of the U.S. Environmental Protection Agency has determined that the publication of this periodical is necessary in the transaction of public business required by law of this Agency. Use of funds for printing this periodical approved by the Director of the Office of Management and Budget February 6, 1973.

Pesticides Abstracts is available on 16 mm microfilm. Additional information can be obtained from the Technical Services Division.

ORDER FORM FOR
PESTICIDES ABSTRACTS

To: Superintendent of Documents
Government Printing Office
Washington, D.C. 20402

Please enter my subscription for Pesticides Abstracts. I am enclosing Money Order ☐ Check ☐ for this subscription.
($18.25 a year; $4.60 additional for foreign mailing.)

Please address as follows: _____

_____

_____

UNITED STATES GOVERNMENT PRINTING OFFICE, WASHINGTON, D.C. 1968

For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402. Subscription price, domestic, $18.25 a year, $4.60 additional for foreign mailing. Price for a single copy of this issue is $1.40, Index issue $2.35.

U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF PESTICIDE PROGRAMS
Technical Services Division
Rm. E-315, 401 M Street, S.W.
Washington, D.C. 20460

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF HEW

Serials Department
Xerox University Microfilms
300 North Zeeb Road
Ann Arbor, MI 48106 EPA 18-1

If you do not desire to continue receiving this publication, please CHECK HERE ☐; cut off this label and return it to the above address. Your name will then be removed from the mailing list.